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A routine refraction with trial lenses should be done in order to arrive at the best possible correction. Each eye is tested separately. The correction is placed in the rear of the trial scope. With the trial correction incorporated in the scope it is often possible to better the correction because the increased magnification permits the patient to better evaluate small changes in correction or axis.

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Each eye is tested separately for both distance and near. When the examination is completed it is necessary to decide whether the eyes are to be used binocularly for near or for distance. It has proven best to set the visual axes parallel, which means the scope will be used binocularly for distance. The patient then uses one eye at a time for reading. The other eye being occluded by a frosted cap.

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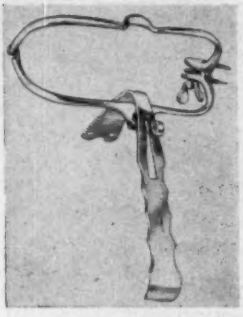

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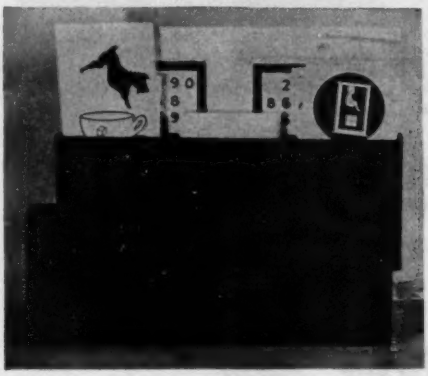

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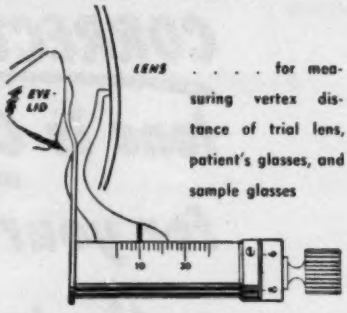

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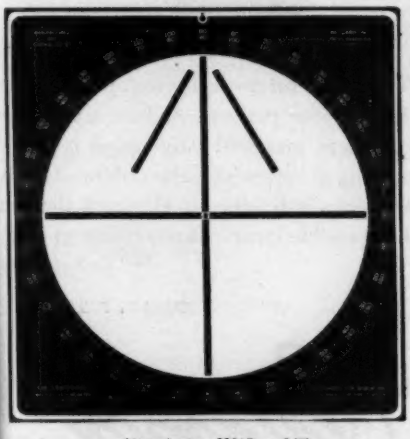

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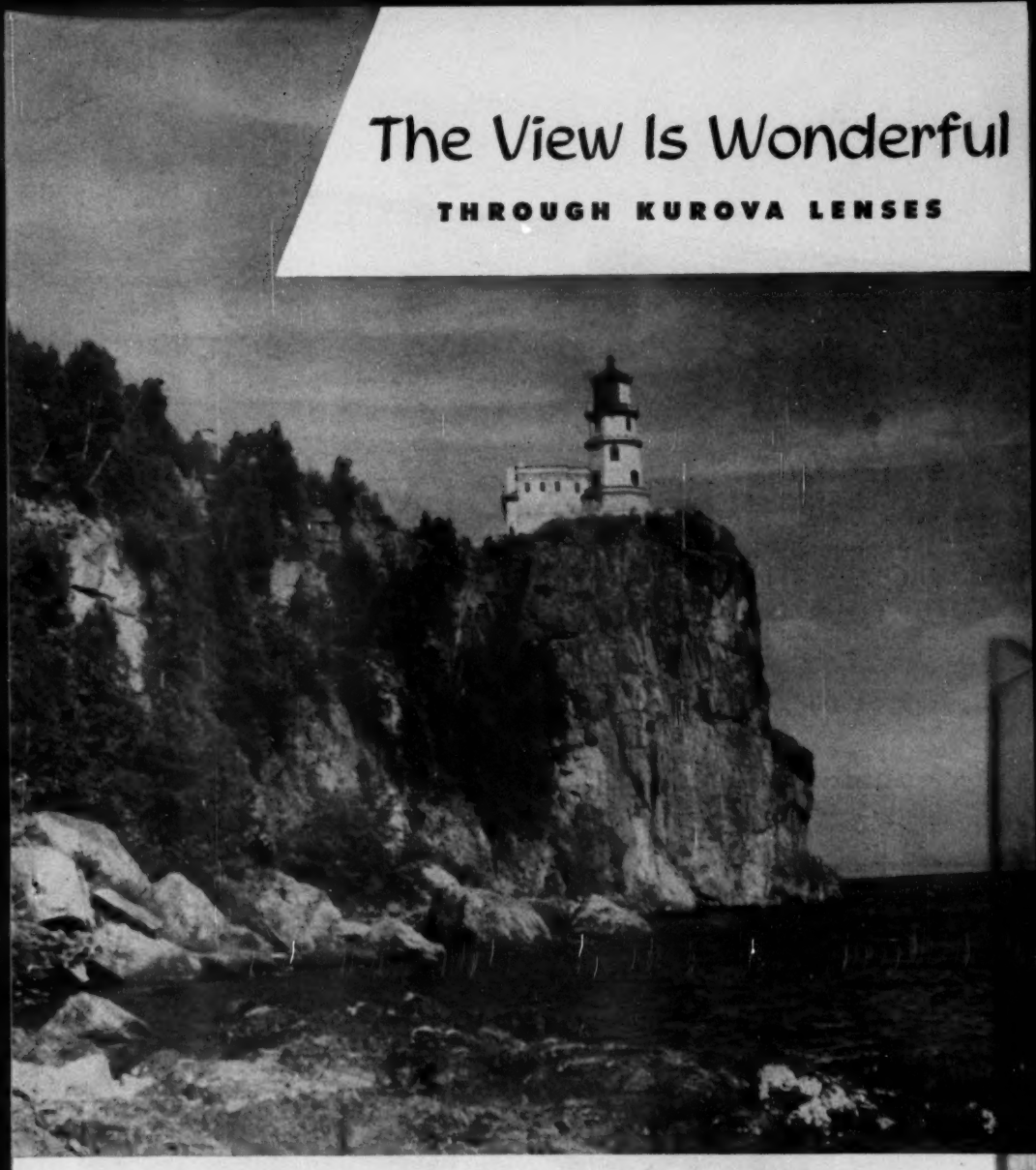

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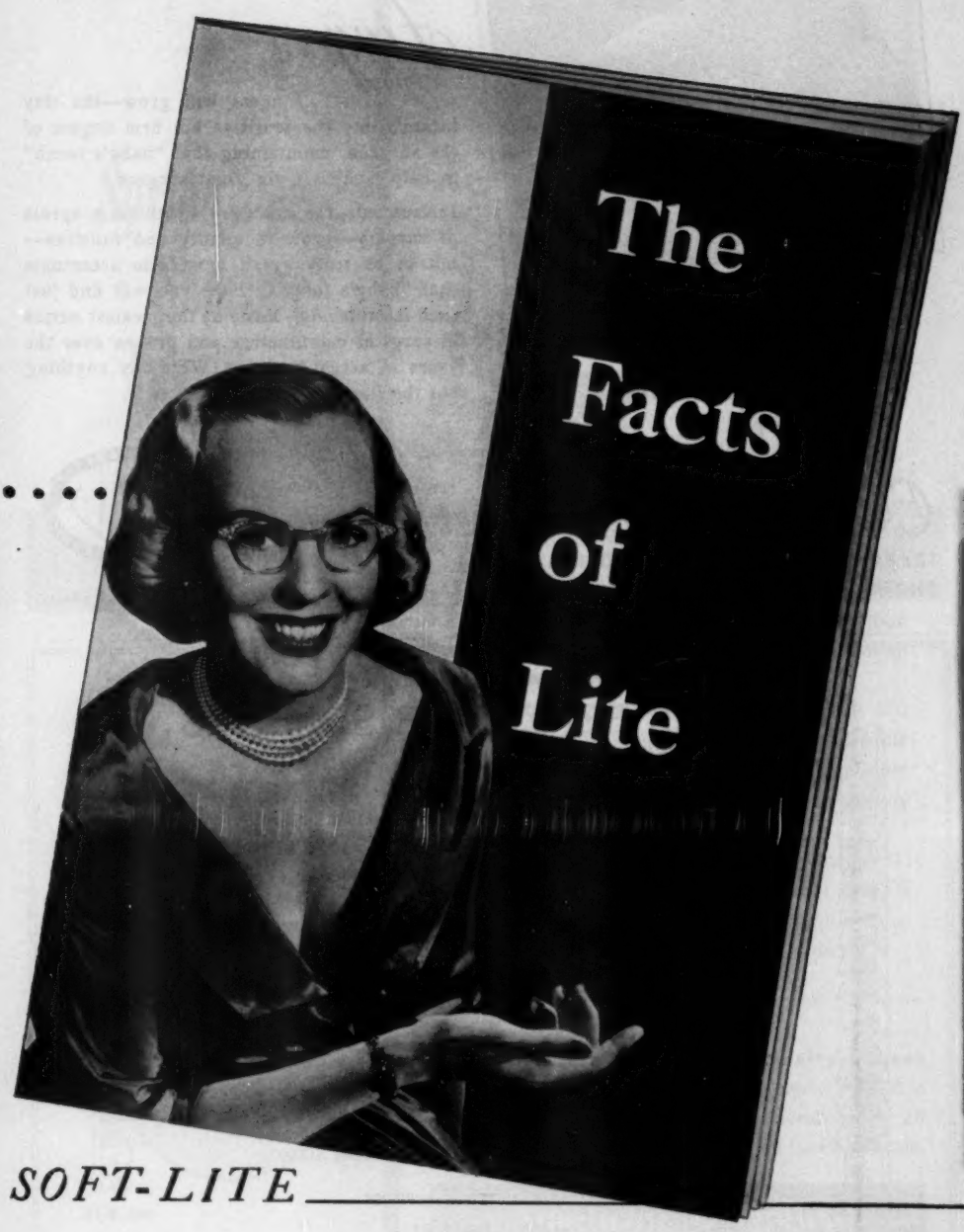

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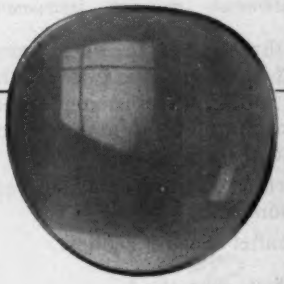

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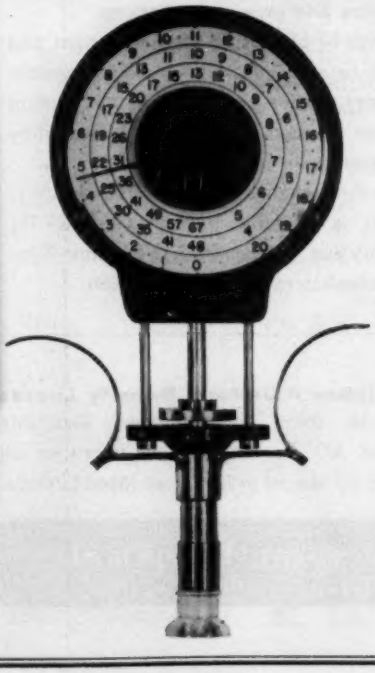

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Editorial

The most difficult task of the Editorial Board of a scientific periodical is the rejection of those manuscripts which are judged unsuitable for publication. Suitability is a rather vague characterization, based on many different and often diverse qualities. During the last two years one reason for rejection has occurred more frequently than any other. More than two thirds of the rejected manuscripts were returned to their authors because of failure to provide adequate controls from which the reader could judge for himself whether the author's conclusions merit acceptance.

It seems strange that this should be so, in view of the fact that most contributors to our journals have had excellent scientific training. However, ophthalmologists are not alone in failing to provide adequate controls in their papers. Ross [1] has recently called attention to the fact that 45 per cent of articles published by a group of leading medical periodicals in the country had no control group of cases reported from which one could compare the effect of a recommended therapy. Eighteen per cent used a control that was considered inadequate, and only 27 per cent presented what Ross considered to be adequate controls. I quote from Ross's article:

> On reading current medical journals, one is impressed by the number of articles which extol one or another form of therapy, but which frequently lack proof of scientific merit. The great number and variety of incompletely tested treatments that are proposed as being of value, often for the same disease process, stand in bold contrast to the few which are rigidly tested according to accepted scientific criteria. . . .
>
> Controls are untreated patients, essential to any scientific experiment because the variable factor being measured must be compared to constants or to known variables. The course of a disease process following a form of therapy must be shown to be improved over the natural course of the disease or over other standard treatment, and in a statistically significant number of cases. This standard seems clearly necessary for an adequate evaluation, but my data indicate that it is often misused or omitted. Thus, there creep into medical literature many treatments which are quoted and requoted and finally unequivocally accepted, but which could not stand rigid scientific scrutiny.

It must be recognized that there are frequent occasions when adequate controls cannot be secured. The author should be the first to point this out, however, and temper his conclusions accordingly. In evaluating any form of treatment, a control group of untreated patients, or patients who are treated by some other means, is absolutely necessary.

1. Ross, O. B., Jr.: Use of Controls in Medical Research, J. A. M. A. **145**:72-74 (Jan. 13) 1951.

Effective control is the key to the entire experimental procedure. It is essential for the accuracy of the conclusions. Without proper control we cannot be certain that the causal nexus which we seek to establish is a real one. When an experiment has been conducted without good controls, we cannot know whether an observed effect is actually attributable to the hypothetical cause or to some other equally uncontrolled factor. We cannot tell whether the result would have been the same in the absence of any one of the factors.[2]

It is hoped that in the future all authors will include adequate controls in papers dealing with the effects of treatment. Acceptance of the paper will be much more likely, and its value to our readers markedly enhanced. In some cases this procedure will possibly convince the author that his conclusions were erroneous.

FRANCIS HEED ADLER, M.D.

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2. Greenwood, E.: Experimental Sociology: A Study in Method, New York, King's Crown Press, 1945.

HARADA'S DISEASE AND VOGT-KOYANAGI SYNDROME

Uveoencephalitis

ALEXANDER R. COWPER, M.D.
BUFFALO

THE PURPOSE of this paper is to present a case of Harada's disease, to emphasize certain unusual clinical findings and to point out that the differences between the Vogt-Koyanagi syndrome and Harada's disease are superficial and do not warrant classification of the two as separate disorders.

Harada's disease, like the Vogt-Koyanagi syndrome, is a severe nontraumatic bilateral uveitis, usually with one or more associated features; namely, alopecia, poliosis, vitiligo and dysacousia. In a large percentage of the cases reported belonging to both groups, there is a prodromal meningismal episode which may be marked by nausea, emesis, fever, severe headache, drowsiness, vertigo, pleocytosis and occasionally other neurological findings. While the incidence of both diseases is higher among the heavily pigmented races and between the ages of 20 and 50, it does not seem related to sex, geographic location or economic factors. The course of each illness is chronic, running for many months; the prognosis, with respect to vision, is serious. In neither condition is the etiological agent known.

Attempts to classify the two as separate entities depend mainly on three clinical findings: the presence or absence of bilateral retinal detachments, marked anterior uveal involvement and alopecia with poliosis. Retinal separations and absent or low grade anterior uveal involvement supposedly typify the Harada group, while alopecia and poliosis, sometimes present in Harada's disease, are claimed to be invariable features of the Vogt-Koyanagi syndrome.

When dealing with two conditions possessing in common such a bizarre combination of seemingly unrelated signs and symptoms, it is only natural to look for differential features so striking and inconsistent with one another as to be unexplainable by one cause. When one finds on reviewing the literature that two of the three differential points are not valid and that the third, retinal detachment, is probably only secondary to an acute uveitis, a process underlying both conditions, the division between the two seems arbitrary, indeed. The practice of regarding them as separate disorders has led to much confusion in the literature and is attributable to the tendency among writers to repeat without critical examination assertions made by early investigators on the basis of insufficiently large case series.

The following case is presented for several reasons. If Harada's disease is actually a separate syndrome, it is an exceptionally rare one of which fewer than 10 cases have been reported in the English literature. If the two conditions, however, are simply clinical variations of the same underlying process, the case is

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interesting as another example of one possessing supposedly pathognomonic features of each. It is the only case on record, whether of the Vogt-Koyanagi syndrome or Harada's disease, with an electroencephalogram suggesting diffuse cerebral involvement. Finally, a complication developed which has been described by only five other writers, namely, bilateral scleritis, associated here as in no other case reported with transitory staphylomas.

REPORT OF A CASE

E. M., a 27 year old Negro woman, was hospitalized on Feb. 13, 1950. During the preceding 10 days there had been malaise, severe diffuse headache, most intense frontally, and deep orbital pain accompanied with intense photophobia, redness of the eyes and bilateral loss of vision. Past and family histories were noncontributory. Except for nocturia, two and three times per night, and irregular vaginal bleeding, both of several months' duration, the functional inquiry revealed an essentially normal condition. Significant findings on physical examination were limited to the eyes and to a moderate elevation of temperature, 101.6 F. orally. Neurological (extraocular) and otorhinolaryngological consultations yielded nothing significant. Gynecologic examination led to the diagnosis of cystic cervicitis.

Both eyes showed an intense diffuse bulbar injection, superficial and deep, with acute blepharospasm and photophobia. Vision was confined to light perception in each eye. Pupillary reactions were not remarkable, and ocular tensions were within normal limits. Motility was difficult to evaluate because of the photophobia, but the patient appeared unable to follow a light in any direction and could not elevate her gaze on command. Voluntary movements were almost totally uncoordinated, with a tendency toward a marked divergent strabismus, the deviation angle of which changed constantly even in the same fields of gaze. Pronounced tenderness was elicited on pressure over the ciliary region. Although no corneal precipitates were visible, there was a deep aqueous haze obscuring the irises, which appeared muddy and edematous, while the cloudiness of the media permitted only a dim view of the retinas, which were completely separated as bullous detachments and were visible only with + 10 D. lenses.

During the first weeks of hospitalization the patient continued to complain bitterly of headache, deep orbital pain and feverishness in the night. Actually her temperature rose daily in the late afternoon to between 99 and 101.5 F., the peaks gradually subsiding two months after admission. Some three days after admission urinary retention developed, and it was necessary to catheterize the patient for the following week. No previous episodes of this type had occurred, and no local cause of the obstruction could be found. About one month after the onset of her illness, the patient complained for the first time of impaired hearing. This did not clear for several weeks. About two months after the onset those symptoms not directly related to the eyes had completely disappeared. She no longer complained of malaise, headache, orbital pains or hearing impairment. The ocular movements were normal, and the temperature elevations were only slight and occasional. At about this time, however, numerous granulomatous corneal precipitates appeared bilaterally. Bluish crescentic discolorations, 1 and 2 mm. peripheral to and concentric with the upper halves of each limbus, tending to protrude slightly above the surrounding sclera, indicated a complicating scleritis with staphylomas. The tension in each eye had risen to 40 mm., Schiøtz. There seemed to be a slight backward displacement of both lenses, now sparsely covered with pigment deposits, and the chambers were abnormally deep. No posterior synechias developed, however, and the pupillary reflexes were unimpaired. Otherwise, the ocular status remained unchanged. The retinas were faintly visible not far posterior to the lenses. Rapidly controlled with administration of 10 per cent solution of furtrethonium (furmethide®) iodide three times a day, the secondary glaucoma did not recur when the medication was discontinued one month later. At this time, May 9, three months after admission, the patient was discharged, and the only change in her ocular condition was some decrease in the bulbar injection and improvement of vision to perception of hand movements. Treatment had consisted of mydriatics until the furtrethonium iodide was substituted because of the glaucoma, while, systemically, penicillin and streptomycin were used in the early stages until it was apparent that they were having no effect on the course of her illness.

Laboratory Findings.—Repeated urinalyses gave essentially normal results. The hemoglobin concentration was 11.5 Gm. The white blood cell count was 7,880, with a differential count of 10 stab forms, 77 segmented forms, 13 lymphocytes and 10 monocytes. Subsequent differential counts were within normal limits. Blood chemical values, including those for urea nitrogen, proteins, fasting glucose, cholesterol, nonprotein nitrogen and chlorides, were not remarkable. The blood Wassermann test and the test for heterophil antibodies had negative reactions. No cold agglutinins were found in the serum. The corrected sedimentation rate on March 13 was 50 mm. per hour. A fungus skin test series, including coccidioidin, histoplasmin, Frei and Brucella abortus tests, had negative results. Tests with old tuberculin in 1:10,000 and 1:1,000 dilutions gave no reactions.

Spinal taps showed normal dynamic pressure with no elevation. On March 2 there was a pleocytosis, the count being 160 cells per cubic millimeter, with 60 per cent lymphocytes and 40 per cent polymorphonuclear cells. No pathogenic organisms were found on smear or culture. The Pandy and Wassermann reactions were negative. The reaction to the colloidal gold test, 5557888876, was interpreted as an abnormal second zone, B type syphilitic curve. On March 7 and subsequently, spinal fluid cell counts were within normal limits. Roentgenograms of the skull, chest and sinuses revealed no pathological changes.

The electroencephalogram showed considerable fast activity (20 to 30 per second frequency) in all areas but was least prominent in the occipital areas, where occasional six to 10 per second waves were seen. There were rare spikes, which in the right frontal temporal and frontal parietal areas showed phase reversal. The impression was an abnormal electroencephalogram displaying rather diffuse disturbance, most pronounced in the right frontal area.

One month after her discharge and four months after the onset of her illness, a mild diffuse bulbar injection remained. The retinas were located more posteriorly but were still detached. Vitreous haze and aqueous beam were less marked so that the disks could be dimly seen, and the corneal precipitates had decreased in number and size. A purplish crescentic discoloration marked the site of the scleritis, but there was no longer any elevation. Vision remained at perception of hand movements.

Some two months later the picture was unchanged except for three fluffy iris nodules, about 1 mm. in size, located halfway between the pupillary margin and periphery of the iris of the right eye. The normal iris pattern had been obliterated. Transillumination revealed marked generalized depigmentation, enabling one to see faintly the fundus reflex through the scleral coat. Ophthalmoscopic examination showed a further settling back of both retinas, although still detached, and a grayish spotty depigmentation giving a mottled effect, with tiny scattered pigment deposits. A few small black opacities were seen in the still somewhat hazy vitreous. Both disks were gray with ill defined borders. Tension was 14 mm. in each eye, and vision had improved to ability to count fingers.

HISTORY

Vogt[1] is given credit for first describing, in 1906, bilateral uveitis with pigmentary changes as a separate syndrome. He commented, however, only on the associated poliosis. This was first noted to occur in sympathetic ophthalmia by Schenkl[2] in 1873 and 20 years later was described for the first time in association with a nontraumatic bilateral uveitis by Hutchinson.[3] In the latter part of the last century dysacousia was observed in a case of sympathetic ophthalmia by Snellen[4] and later by Komoto[5] and Cramer.[6] In 1910 vitiligo was first reported in association with ocular disease by Gilbert.[7]

1. Vogt, A.: Klin. Monatsbl. f. Augenh. **44**:228, 1906.
2. Schenkl, A.: Arch. f. Dermat. u. Syph. **5**:136, 1873.
3. Hutchinson, J.: Arch. Surg., London **4**:357, 1892-1893.
4. Snellen, H.: Tr. Internat. M. Cong. London **3**:31, 1881.
5. Komoto, J.: Klin. Monatsbl. f. Augenh. **50**:129, 1912.
6. Cramer, E.: Klin. Monatsbl. f. Augenh. **51**:205, 1913.
7. Gilbert, W.: Klin. Monatsbl. f. Augenh. **48**:24, 1910.

It was not until 1929 that alopecia and dysacousia were added to the syndrome by Koyanagi,[8] who presented the first extensive review of the literature, describing six cases of his own and 10 of other Japanese authors. Babel [9] suggested the name Vogt-Koyanagi syndrome, defining it as a bilateral nontraumatic uveitis with any or all of the associated signs—poliosis, vitiligo, alopecia and dysacousia. In his review of 19 cases in 1931, the first in English, Parker [10] pointed out its resemblance to sympathetic ophthalmia. In 1926 Harada [11] described 10 cases of his own as examples of a separate entity complicated by retinal detachment. Subsequently, "Harada's disease" was reported for the first time in Europe by Salus (1932) [12] and in English by Rados (1940),[13] who attempted to differentiate the two conditions on the basis of retinal detachment, anterior or posterior uveal involvement and alopecia with poliosis.

Although a total of more than 50 cases of the Vogt-Koyanagi syndrome has been reported in the literature, not more than 10 cases of Harada's disease have been described in the English literature, including those of Rados, Givner,[14] Walsh [15] and Bruno and McPherson.[16]

CLINICAL PICTURE

The severe, prolonged bilateral nontraumatic uveitis is, of course, the essential feature of both conditions. Preceding this by one or more weeks in well over half of the reported cases of both syndromes is a prodromal meningismal episode, with severe headache, deep orbital pain, drowsiness, vertigo, fever, nausea and emesis. Finally, from several weeks to months following the onset, as the uveitis gradually subsides, the changes in the skin and hair systems occur, i. e., poliosis, vitiligo and alopecia. Dysacousia, if it appears, is a relatively early symptom manifesting itself not long after the onset of the uveitis, or even, occasionally, in the prodromal stage. Likewise, the retinal detachments of Harada's disease, which may be partial or complete, develop during the first few weeks following the onset of the uveitis.

Thus, the illness may be divided into three stages: the meningeal, the ophthalmic and, finally, the convalescent stage, when the associated signs usually appear. Both the dysacousia and the retinal detachments may be regarded as part of the second stage. During the first and early second stages, which overlap, pleocytosis is not an uncommon finding.

Complications include secondary glaucoma, phthisis bulbi, retinal detachments and cataracts; those, in other words, which may follow any severe uveitis. Complete recovery may occur, especially, it is thought, in the Harada group in

8. Koyanagi, Y.: Klin. Monatsbl. f. Augenh. **82**:194, 1929.

9. Babel, J.: Schweiz. med. Wchnschr. **69**:1136, 1939.

10. Parker, W. R.: Am. J. Ophth. **14**:577, 1931.

11. Harada, Y.: Nippon Gankiva Gankukwai Zasshi **30**:356, 1926.

12. Salus, R.: Klin. Monatsbl. f. Augenh. **89**:84, 1932.

13. Rados, A.: Bilateral Uveitis Associated with Detachment of the Retina (Harada's Disease), Arch. Ophth. **23**:534 (March) 1940.

14. Givner, I.: Bilateral Uveitis, Poliosis and Retinal Detachment with Recovery: Report of Case, Arch. Ophth. **30**:331 (Sept.) 1943.

15. Walsh, F. B.: Clinical Neuro-Ophthalmology, Baltimore, The Williams & Wilkins Company, 1946, pp. 548-555.

16. Bruno, M. G., and McPherson, S.: Am. J. Ophth. **32**:513, 1949.

which retinal reattachment develops oftener than not. In Parker's series of 29 patients with the Vogt-Koyanagi syndrome [17] 30 per cent recovered vision of 6/12 or better. On the other hand, in 32 of Rosen's 47 cases [18] there was a "malignant terminus."

The meningismal, or first, stage is claimed to be present in 90 per cent of the cases of Harada's disease and in about 50 per cent of those of the Vogt-Koyanagi group.[19] Its severity varies. Positive Kernig and Brudzinski signs with stiffness of the neck have been found. In a case of Magitot and Dubois-Poulsen [20] the patient was delirious in the early stages; in another reported on by the same authors, there was a sudden loss of consciousness at the onset and later paresis of the right sixth nerve developed. Schiff-Wertheimer [21] observed, in connection with another patient she reported on, a 5½ year old child who had hemiparesis, slight aphasia and a unilateral convergent squint. In those cases in which neurological findings have not been noted, it may be for the reason that they had either subsided or been overlooked. Certainly in both conditions a lymphocytic pleocytosis, with transitory elevation in spinal fluid pressure, has been found on numerous occasions. Indeed, for this reason Rubino [19] contends that Harada's disease should be regarded as basically a uveomeningitis, the anatomicoclinical basis of which is probably found in the embryogenetic, morphological and functional homolog between the uveal membranes and the leptomeninges. In Rosen's comprehensive summary of the literature on the Vogt-Koyanagi syndrome in 1945,[18] he pointed out that in 20 of the 47 cases there was initial cephalalgia.

Shortly after the appearance of the meningeal symptoms, sometimes coinciding with it, the uveitis sets in. The onset may be gradual, with only cells in the aqueous,[22] or fulminating, with intense bulbar injection, turbidity of the media and numerous granulomatous corneal precipitates. When the fundus can be seen through the vitreous opacities, diffuse choroiditis and optic neuritis with pronounced surrounding retinal edema are often noted. In the majority of cases, however, the fundi are soon lost to view. The retinal separations, as might be expected, occur at the height of the uveitis, early in the second stage of the disease, while in a good percentage of the cases reattachment follows during subsequent months as the uveitis, often with remissions and exacerbations, tends to subside. Total clearing of the uveitis may not occur for a year or more.

In the second stage, as a rule, the dysacousia—deafness and/or tinnitus—manifests itself. Reported in 54 per cent of Rosen's cases, it is thought to be of the central nervous system or labyrinthine type,[17] is generally bilateral and tends to disappear in weeks or months. Scleritis has been reported by several authors, usually in a discrete and nodular form, but sometimes occurring more diffusely.

Vitiligo, poliosis and alopecia develop most commonly in the first three months, when the uveitis has begun to abate, although they have been reported to appear

17. Parker, W. R.: Severe Uveitis with Associated Alopecia, Poliosis, Vitiligo and Deafness: A Second Review of the Published Records, Arch. Ophth. **24**:439 (Sept.) 1940.

18. Rosen, E.: Uveitis, with Poliosis, Vitiligo, Alopecia and Dysacousia (Vogt-Koyanagi Syndrome), Arch. Ophth. **33**:281 (April) 1945.

19. Rubino, A.: Ophthalmologica **101**:321, 1941.

20. Magitot, A., and Dubois-Poulsen, A.: Bull. Soc. d'opht. Paris **51**:223, 1939.

21. Schiff-Wertheimer, S., in discussion on Magitot and DuBois-Poulsen,[20] p. 227.

22. Laval, J.: Am. J. Ophth. **21**:536, 1946.

as late as nine months following the onset. In some cases none of these signs is present, while in the majority one or two are absent. In Rosen's series poliosis occurred in 90 per cent, alopecia in 73 per cent and vitiligo in 63 per cent. The alopecia is of the areata type and eventually disappears. The poliosis and vitiligo may involve any part of the body, are often symmetrical and are most apt to occur on the head, neck and trunk. These changes, also, generally disappear.

During the third stage, coinciding with the disturbance in skin and hair systems, the uveitis gradually diminishes. The eyes present an appearance similar to that following any severe uveitis except for more extensive and more marked depigmentation. White areas, giving a mottled effect, may be scattered throughout the fundi, interspersed with pigment deposits. Posterior synechias binding down the iris may give evidence of the previous iridocyclitis, and secondary glaucoma not infrequently intervenes. Davies [23] reports that in his series of 21 patients with the Vogt-Koyanagi syndrome 46 per cent showed hypertension and 10 per cent hypotension.

ETIOLOGICAL FACTORS

It is not remarkable that a common etiological agent has been suggested for the Vogt-Koyanagi syndrome, Harada's disease and sympathetic ophthalmia; all three are syndromes of bilateral uveitis in which any one or a number of the same associated signs may be seen.

Of all the causes of uveitis in general, those established most frequently are syphilis, tuberculosis and foci of infection. None is thought to be involved in the conditions in question. Syphilis and tuberculosis, at one time incriminated, have been excluded by laboratory and immunologic tests. Foci of infection, for once, have not been seriously considered.

To explain the widespread findings, three theories have been advanced—endocrine, allergic and viral. Exponents of the endocrine hypothesis point out the part the endocrine system is recognized to play in pigmentary disturbances in Addison's disease and eunuchoid states, as well as in the occasional choroiditis seen with ovarian dysfunction. Currently, the theory receives little support and emphasis is placed on the allergic and viral hypotheses.

Elschnig [24] was the first to propose allergy as a basis for uveitis associated with pigmentary changes. Sympathetic ophthalmia, according to him, results from the release through injury of uveal pigment with antigenic properties and from the formation of organ-specific antibodies, the interaction between which produces anaphylactic pigment destruction. Cramer [6] applied the theory to the Vogt-Koyanagi syndrome, explaining the deafness as due to the presence of pigment in the basilar membranes of the labyrinths. Koyanagi,[8] who favored a virus origin, rejected this explanation on clinical grounds, pointing out that the dysacousia often occurs too soon after the onset of the illness to allow time for antibody formation. Anatomic evidence is lacking, in that pigmentation of the basilar membrane does not occur normally in man. Laboratory tests cast further doubt on the theory so far as in several cases of uveoencephalitis, in which tests were made for sensitivity

23. Davies, W. S.: Uveitis with Associated Alopecia and Poliosis, Vitiligo and Deafness, Arch. Ophth. **14**:239 (Aug.) 1935.

24. Elschnig, A.: Arch. f. Ophth. **76**:509, 1910.

to uveal pigment, the results in all but one [25] were negative or doubtful. Other questions arise—for example, how to explain the prodromal encephalitis in terms of allergic response, and why it is that other forms of uveitis do not produce similar anaphylactic reactions.

The evidence for a viral pathogenesis, on the other hand, is impressive. As early as 1910 Gilbert [7] suggested the herpes zoster virus as the cause in his case. Both vitiligo and alopecia have been observed in herpes zoster. While attempts to demonstrate a virus to be an etiological factor have frequently failed, the work of several investigators points in this direction. Vitreous fluid taken from a patient with Harada's disease by Takahashi [26] was injected intracisternally in rabbits and produced in two of five animals a descending neuritis and uveitis. The inoculation of the cerebrospinal fluid of the patient into the vitreous of rabbits caused inflammation in one of four animals. Confirmatory work has been done by Tagami,[27] Friedenwald [28] and Malbran and Muhlmann.[29] The last two injected spinal fluid from their patients into the vitreous of rabbits, with a resulting intraocular inflammation, which, 12 to 16 days later, appeared in the untreated eye.

Apropos of the postulated infective nature of the disease, it is interesting to note that the literature contains only one case in which another person in contact with the patient acquired bilateral uveitis.[12]

Various explanations have been offered for the disturbance in skin and hair systems. Some ascribe it to a virus with melanotropic or neurotropic properties, pointing out that vitiligo, alopecia and poliosis are all classifiable as dermatoneuroses. Rubino,[19] stressing the meningoencephalitic aspect of the disorders, argued that involvement of the pars intermedia of the pituitary, known to be the pigment-regulating center of the body, might lead to the pigmentary changes. Wexler [30] regarded the process as primarily one of depigmentation which, with reference to the eyes, in his case was extreme.

After an exhaustive discussion of the Vogt-Koyanagi syndrome, Hague [31] concludes that

> to me it seems possible that a lesion involving the fiber tract from the supraoptic region to the hypophysis produces the alopecia areata, the poliosis and the dermal vitiligo and that the profound disturbances of the pigment structures in the eye may be responsible for the severe inflammatory process which follows, somewhat as a melanotic sarcoma of the choroid may produce intraocular inflammation. . . .

The dysacousia he explains as possibly the effect of the lesion on Gudden's commissure, a part of the auditory system of the forebrain.

It has already been mentioned that in many respects sympathetic ophthalmia resembles the Vogt-Koyanagi syndrome and Harada's disease. This has been

25. Rones, B.: Uveitis with Dysacousia, Alopecia and Poliosis, Arch. Ophth. **7**:847 (June) 1932.

26. Takahashi, M.: Acta Soc. ophth. japon. **34**:506, 1930.

27. Tagami, R.: Acta Soc. ophth. japon. **35**:1289, 1931.

28. Friedenwald, J. S., and McKee, C. M.: Am. J. Ophth. **21**:723, 1938.

29. Malbran, J., and Muhlmann, V.: Cong. argent. de oftal. (1936) **2**:194, 1938.

30. Wexler, O.: Ocular Depigmentation Accompanying Generalized Vitiligo, Arch. Ophth. **57**:393, 1928.

31. Hague, E. B.: Uveitis; Dysacousia; Alopecia; Poliosis, and Vitiligo: A Theory as to Cause, Arch. Ophth. **31**:520 (June) 1944.

underlined by recent studies showing that pleocytosis, with other meningeal signs, is not uncommon in sympathetic ophthalmia.[32] Histopathological examination, again, illustrates the close relationship, since all three conditions are manifest in the eye by a chronic granulomatous inflammatory process with conspicuous disorganization of chromatophores.

COMMENT ON CASE

Among the features of special interest in the present case are, first, the severity of the meningismal stage, which showed the unusual findings of extraocular muscle imbalance and, possibly, a neurogenic bladder. It is the only case on record with a positive electroencephalogram, the nature of which points to diffuse cerebral involvement, such as found in meningoencephalitis.

Second, the case represents a form intermediate between Harada's disease and the Vogt-Koyanagi syndrome, with its bilateral retinal detachments in the presence of a fulminating anterior uveitis, as evidenced by the intense and prolonged bulbar injection, by the thick coating of corneal precipitates and, particularly, by a complicating bilateral scleritis. The staphylomas secondary to the scleritis have not been reported in any other case. Third, the poor prognosis concerning vision, which in this case is exceptionally bad in view of continued retinal separations eight months following the onset, is said to be characteristic of the Vogt-Koyanagi syndrome, not Harada's disease.

Finally, a number of clinical findings, only occasionally mentioned in the literature, were present in this case, including fluffy iris nodules, extreme depigmentation of the eyes enabling one to see the fundus reflex through the scleral coat, strangely enough unaccompanied in this case with extraocular pigment changes, a secondary glaucoma successfully treated with miotics and, lastly, a backward displacement of iris-lens diaphragm, with deepening of the chambers, perhaps secondary to the staphylomas.

WHY TWO ENTITIES?

Separate classification of bilateral nontraumatic uveitis as the Vogt-Koyanagi syndrome and Harada's disease probably depends in large part on the observations of two early writers, Parker [33] and Rados.[34] Recent studies have invalidated the three fundamental differential points—the presence or absence of retinal detachment, anterior uveal involvement and alopecia with poliosis.

Parker [10] himself states regarding Vogt-Koyanagi syndrome that "the incidence of neuroretinitis and detachment of the retina may be far from correct as a satisfactory fundus examination is impossible in many cases," an observation made by many subsequent authors. In Rosen's study of 47 cases of Vogt-Koyanagi syndrome, seven were found to show retinal detachment.

Parker's two reviews of the literature on the Vogt-Koyanagi syndrome are probably the source of the often repeated statement that alopecia and poliosis

32. Coreelle, L.: Brit. J. Ophth. **31**:366, 1947.

33. Parker.[10] Parker.[17]

34. Rados.[18] Rados, A.: Bilateral Uveitis with Associated Detachment of the Retina, Arch. Ophth. **26**:543 (Oct.) 1941.

invariably accompany the Vogt-Koyanagi syndrome. Even Duke-Elder [35] asserts that alopecia is always present. Several later case series, however, have not borne this out. Carrasquillo [36] observed alopecia to be present in only 53 per cent and poliosis in 82 per cent of his 29 cases, while Rosen set the incidence at 73 and 90 per cent, respectively.

Nor can any hard and fast rule be made regarding anterior uveal involvement. Rados [13] wrote that Harada's disease is "characterized by either lack of, or low grade, inflammatory signs in the anterior segment of the uvea," an assertion appearing since in the literature with monotonous regularity. The first patient with Harada's disease reported on in Europe by Salus [12] displayed severe anterior uveitis, and of the fewer than 10 patients so far reported on in the English literature, at least half, including the one under discussion, demonstrated extensive involvement of the anterior segments.

Still, it may well be true that in the Vogt-Koyanagi group anterior uveal inflammation is present in a higher percentage of cases than in the Harada group. If so, it would serve to explain, first, what is probably a lower incidence of retinal detachment in the Vogt-Koyanagi group, since retinal detachment, is, of course, far less apt to occur as a complication in an iridocyclitis than in an acute exudative choroiditis, and, second, the poorer prognosis with respect to vision associated with the Vogt-Koyanagi syndrome. It seems likely that in any large series of cases of uveitis divided into one group with predominantly anterior uveal involvement and into another with predominantly posterior uveal involvement, the former would show a higher incidence of serious visual impairment. As against the danger of macular involvement in posterior uveitis, there is in anterior uveitis the greater danger of such complications, each a threat to vision, as secondary glaucoma, secondary cataract, hypotonia with phthisis bulbi and inflammatory membranes obstructing the pupillary opening.

In any event, whether or not in one the seat of pathological change is primarily anterior and in the other primarily posterior, it is difficult to conceive of a more unsatisfactory basis for the separate classification of two disorders, in both of which the causative agent is unknown and in each of which such a widely distributed and bizarre constellation of manifestations is found.

And, as if this were not enough, it is common knowledge that in those inflammations of the uvea in which the etiological agent is considered established, in the syphilitic and tuberculous uveitides, in those due to foci of infection and to allergy—in none of these has any serious attempt been made to diagnose the condition according to whether the anterior or the posterior uvea is involved.

Many authors have adopted the viewpoint of a single cause.[37] There has been an ever increasing demand among recent writers to regard the two conditions as

35. Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1940, vol. 3, pp. 2364-2367.

36. Carrasquillo, H. F.: Uveitis with Poliosis, Vitiligo, Alopecia and Dysacousia (Vogt-Koyanagi Syndrome), Arch. Ophth. **28**:385 (Sept.) 1942.

37. (*a*) Rubino.[19] (*b*) Magitot and Dubois-Poulsen.[20] (*c*) Martinez, L.: An. Soc. mex. de oftal. y oto-rino-laring. **16**:273, 1941. Avalos, E.: Rev. cubana de oto-neuro-oftal. **5**:75, 1936.

simply minor clinical variations in the same underlying process.[38] Kitazawa [39] describes histopathological changes accepted by Matsuoka [40] and Ogawa,[41] supporting the identity of the two disorders, while, in contrast, absolutely no acceptable evidence of a dual cause exists today.

In view of the confusion to which such an arbitrary distinction has led, examples of which abound in the literature, it is suggested that another name be adopted. Martinez [37c] proposed the name Vogt-Harada disease. More descriptive and probably better justified is the term "uveoencephalitis," which conforms to the latest clinical and laboratory findings.

The present case is submitted as another example of an intermediate form of bilateral uveitis combining features which can no longer be looked on as pathognomonic of either the Vogt-Koyanagi syndrome or Harada's disease, but rather as characteristic of the syndrome of uveoencephalitis.

SUMMARY

A case of so-called Harada's disease is presented with an unusually complete symptom complex. Special emphasis is placed on certain outstanding clinical findings, including a positive electroencephalogram, never previously reported. An analysis of the literature leads to the conclusion that instead of two separate entities there is but one. A new name is proposed.

38. Givner.[14] Rosen.[18] Bruno and McPherson.[16] Harley, R. D., and Wedding, E. S.: Am. J. Ophth. **29**:524, 1946.

39. Kitazawa (1927), cited by Magitot and Dubois-Poulsen.[20]

40. Matsuoka, H.: Zentralbl. f. d. ges. Ophth. **30**:520, 1933.

41. Ogawa, A.: Acta Soc. ophth. japon. **38**:68, 1934.

SCOPE AND LIMITATIONS OF ORTHOPTICS IN NONSURGICAL TREATMENT OF OCULAR DEVIATIONS

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WHEN I was invited to speak at this symposium, various topics came to my mind which might profitably be discussed. However, I discarded all specific topics, since it seemed to me that a topic of more general nature would be better suited to a joint gathering of orthopists and ophthalmologists.

I am frequently asked what I really think of orthoptics. I have felt that this question should some time be answered in more elaborate form. This is why I am speaking tonight about the scope and limitations of orthoptics.

Before considering its scope and limitations, one must first agree on a definition of orthoptics. In the broadest sense of the word, every form of nonsurgical treatment of neuromuscular anomalies of the eyes is orthoptic therapy. In this broadest sense the prescription of glasses for the normalization of the convergence-accommodation relationship is a form of orthoptic treatment. If this definition is considered to be too all inclusive, one may say that every treatment of neuromuscular anomalies of the eyes by nonsurgical means, with the exception of the prescription of glasses, constitutes orthoptic treatment.

It is necessary, therefore, to inquire into what constitutes the province of the surgical treatment and what constitutes the province of the nonsurgical treatment of these anomalies. The two provinces are delimited by the general principles governing the treatment of the neuromuscular anomalies of the eyes. According to these principles, surgical treatment has a direct effect only on mechanical conditions, by creating new and normalized anatomic conditions. It can affect innervational conditions only indirectly, so far as these adjust themselves postoperatively to the changed mechanical situation. But, at the same time, surgery is the only means by which a permanent change in the relative position of the eyes can be achieved. Nonsurgical means, on the other hand, can only affect and normalize the innervational conditions, but can never achieve a permanent change in the relative position of the eyes.

Let me cite some characteristic examples. A child with a purely accommodative strabismus may have perfectly straight eyes as long as he wears the proper correction. As soon as the child removes the glasses and strains his accommodation, the strabismus will return. Such a child should not be operated on, but he may

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be given orthoptic exercises to help him keep his eyes straight without glasses. A patient with exotropia may have learned spontaneously or through orthoptic exercises how to keep his eyes straight by a more or less voluntary effort of convergence. His eyes remain straight as long as he exerts this effort. Such a patient may be operated on to change the relative position of his eyes, thus obviating the need for the constant effort of convergence. Finally, in a case of nonaccommodative esotropia, with a large, stable angle of squint, prolonged orthoptic treatment is useless. Only surgery can be expected to reduce the angle of squint in such cases.

There is another general principle which must be recalled. The sensory symptoms in squint do not represent anything qualitatively new in the visual act. The sensory symptoms and reactions which the orthoptist seeks out or tries to remedy are nothing but a pathological exaggeration of essentially physiological elements of binocular vision. Thus, pathological suppression, one of the foremost enemies of the orthoptist, is only an exaggeration of the suppression which is normally present. Similarly, all other sensory symptoms exhibited by patients with strabismus are prepared or preformed in normal binocular vision. I have attempted to show this for anomalous correspondence in a paper which I read before this group at last year's meeting.[1]

On the basis of these two principles—the fundamental principle governing the therapy of patients with strabismus and the principle that all sensory symptoms of patients with strabismus are pathological exaggerations of physiological functions—one may now consider the scope and limitations of orthoptics.

Orthoptics was in earlier days much harmed by exaggerated and unjustified claims. Today such exaggerations are the exception, and good orthoptists sin by understating, rather than overstating, their results. But there still are many detractors and enemies of orthoptics. These enemies may be divided into two groups: those who are enemies from ignorance and those who are enemies because they expected too much of orthoptics and were disappointed. The enemies from ignorance do not know what orthoptics can achieve. Those who are enemies from expecting too much are not aware of the reasonable limitations of orthoptics.

The question is then: What can orthoptic exercises do? What may one expect of orthoptic treatment? In the first place, one may expect of orthoptic treatment that it do away with certain faults in the monocular and binocular visual act. Orthoptic treatment can never create anything new in the visual act, but it can improve or normalize the elements necessary for normal monocular and binocular vision; it can correct pathological exaggerations or pathological deficiencies of these elements. For example, orthoptic exercises can and do improve central fixation and the visual acuity of amblyopic eyes. They can and do combat suppression, that pathological suppression which prevents one eye from taking part in the act of binocular vision in the proper way. Orthoptic exercises can and do, in appropriate cases, remove anomalous retinal correspondence.

When orthoptic treatment has done away with these visual faults, it may then be expected to encourage proper visual habits and to train those visuomotor functions which are trainable. It may be expected, for instance, that in a case of

1. Burian, H. M.: Anomalous Retinal Correspondence: Its Essence and Its Significance in Diagnosis and Treatment, Am. J. Ophth. **34**:237-253 (Feb.) 1951.

accommodative strabismus the patient will be taught how to dissociate accommodation from convergence and thus be enabled to keep his eyes straight, while having a clear image of the object looked at. It may be expected of orthoptic exercises that they increase and improve the fusional amplitudes of a patient. It may be expected of orthoptic exercises that they improve, when necessary, the near point of convergence. Last, but not least, it may be expected that they give comfortable binocular vision to patients with heterophoria who display symptoms.

So much for what orthoptics can be expected to do. What can orthoptics not be expected to do? Here, I wish, first of all, to state that orthoptics cannot "cure" a strabismus. What is meant by this statement? I mean that if a "cure" of strabismus implies the permanent removal of an angle of squint, such a cure may not be expected of orthoptic treatment. I believe that in the past it was thought by certain ophthalmologists and by the vast majority of orthoptists that such a cure by orthoptic means was possible. It appears that this is no longer generally true. It is now more broadly recognized that orthoptic training cannot change the angle of squint permanently, but that it can achieve, as I have pointed out, a change in the angle of squint by transforming a manifest into a latent strabismus, i. e., by transforming a heterotropia into a heterophoria. The strabismus will remain latent, however, only as long as the conditions last which were created by the orthoptic training—for instance, as long as the child keeps convergence and accommodation dissociated, or as long as the patient with intermittent exotropia maintains the compensatory convergence innervation, or as long as the patient's eyes are not dissociated in one way or another. Otherwise, the manifest deviation will return.

More especially where a mechanical or a paralytic condition is the underlying cause of a strabismus, orthoptic exercises are powerless. It is most unfair, and, in fact, a misuse of orthoptics, when, as sometimes occurs, the ophthalmologist operates on a patient, say for esotropia, never for a moment thinking that he ought to send the patient for an orthoptic check-up or for orthoptic treatment, and, after having produced a postoperative exotropia, then sends the patient to the orthoptist, expecting that the orthoptist should now undo what he has done. Such a surgeon is disappointed, and may even lose all faith in orthoptic treatment, because the orthoptist is unable to correct, to cure, the postoperative exotropia caused by excessive weakening of the action of an internal rectus muscle. It is wrong to expect orthoptics to produce a cure in such a case, and it is certainly wrong to condemn orthoptics because it is incapable of curing the condition. The case simply is not in the realm of orthoptics. The same principle applies to other cases of paralysis, more especially to cases in which a vertical component is prominent.

While orthoptic treatment is thus incapable of permanently changing an angle of squint or of correcting deviations in which innervational factors play no, or only a small, role, there is a further thing that orthoptic exercises cannot do which is not as generally understood.

It is impossible to compel some one to fuse who is incapable of fusing, just as it is impossible to compel any one to perceive depth who cannot see stereoscopically. But the orthoptist can encourage fusion by offering optimum stimulus conditions to make fusion possible. This means that the orthoptist can, first of all, remove such faulty monocular or binocular visual habits as poor fixation, suppression, and the like, which may present an obstacle to fusion. It means,

furthermore, that the orthoptist should attempt to offer to each individual patient fusion stimuli which are best suited to his case by selecting targets for the major amblyoscope that are easiest for him to fuse. Beyond this the orthoptist cannot go.

There are many cases which by their nature are not amenable to orthoptic treatment. However, there is no reason to eliminate from such treatment every case which is unsuitable for orthoptic cure. Judiciously applied, orthoptic exercises may be of great benefit even in definitely nonorthoptic cases. One may be fully aware that the strabismus of a child can be cured only by surgical means. Nevertheless, if such visual faults as suppression and anomalous correspondence are removed prior to operation, the patient will be in a much better position to respond to the surgical straightening of his eyes by using them together properly. Preoperative orthoptic treatment, therefore, is much more likely to insure a functional cure following operation than when surgical treatment alone is employed. It goes without saying that postoperative orthoptic treatment is made considerably easier by the reduction of the deviation.

One last point relates, strictly speaking, not to the limitations of orthoptics, but, rather, to the limitations which the ophthalmologist should impose on himself in selecting patients for orthoptic exercises. Not every patient who may be amenable to orthoptic treatment must be so treated. Obviously, treatment should be given in all cases in which it may reasonably be expected that exercises may influence beneficially the manifest deviation. This holds true especially for all cases of latent deviations in which exercises may rid the patient of symptoms. If it is a matter not of reducing a deviation or of giving comfort but of establishing binocular cooperation, judgment should be exerted in the selection of cases. It may be the part of wisdom to leave some patients alone and to rest content with a cosmetic, surgical result. There are both social and medical contraindications to orthoptic treatment. In cases in which the restoration of binocular vision may be expected to be a difficult, long process, the effort expended on the part of the orthoptist, the child and the parents may be all out of proportion to the result achieved. The orthoptist had better devote herself to patients who are more promising or more in need of her help. The child had better employ his time in practicing music or some other useful skill, and the mother had better use her valuable time in caring for her children and her many duties.

With regard to the medical contraindications, nature's way of giving the patient comfortable single vision by means of suppression is often the best and wisest way. For instance, in a case of high anisometropia, the patient would be very likely to exchange comfortable monocular vision for binocular vision of doubtful quality, productive of symptoms, if he were given exercises.

If anywhere, it is in the decision regarding the institution of orthoptic treatment that it is necessary to give individual consideration to every case in all its aspects, ophthalmologic, as well as social and educational.

SUMMARY

Orthoptic treatment can do away with certain visual faults, such as improper fixation, suppression and anomalous correspondence. It can develop proper visual habits and assist in the training or development of certain visuomotor functions. It can give comfortable vision to patients who have binocular vision only at the

expense of ocular comfort. It may also be used in cases which are definitely non-orthoptic, as a valuable adjunct to the surgical therapy, in an effort to insure a functional cure.

What orthoptic training cannot do is to change the angle of squint permanently, that is, "cure" a strabismus or compel some one to fuse or perceive stereopsis who does not have the faculty of sensory fusion or of stereopsis.

Not every patient who may seem to be amenable to orthoptic treatment should automatically be given exercises. The ophthalmologic, social and educational indications and contraindications must be carefully weighed in each case.

These, in brief, are the scope and limitations of orthoptics. I do not doubt that if these were clearly recognized by all concerned it would make for even happier and more widespread cooperation between orthoptists and ophthalmologists than exists today.

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EXPERIMENTAL CATARACT PRODUCED BY THREE CENTIMETER PULSED MICROWAVE IRRADIATIONS

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THE DEVELOPMENT of cataract as a result of electromagnetic radiations is well established. Formerly it was supposed that this effect was unique to the action of infra-red, ultraviolet and roentgen radiations, although as early as 1926 in a survey of work in these fields Duke-Elder [1] suggested that other portions of the spectrum, if of sufficient magnitude, might produce cataract. On the other hand, as late as 1944 it was the opinion of Bellows,[2] basing his views on the work of Legge [3] and of Hartridge and Hill,[4] that electromagnetic waves longer than 20,000 angstrom units would be absorbed completely by the cornea.

In 1948 it was demonstrated by Richardson, Duane and Hines [5] that lenticular opacities could be produced by continuous microwave radiations of 12.25 cm. wavelength. These observations were confirmed by the contemporary work of Daily, Wakim, Herrick and Parkhill,[6] which was completed later in 1948. The following year Salisbury, Clark and Hines [7] in a technical article attributed the damage to a ready transmission through the cornea and a high absorption by the lens of microwaves 10 to 12 cm. in wavelength. In 1950 Cogan [8] suggested that the most

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1. Duke-Elder, W. S.: Pathological Action of Light upon the Eye, Lancet **1**:1188 (June 19) 1926.

2. Bellows, J. G.: Cataract and Anomalies of the Lens, St. Louis, C. V. Mosby Company, 1944, chap. 7.

3. Legge, T. M., cited by Robinson, W.: Glass-Workers' Cataract, Ophthalmoscope **13**: 545, 1915.

4. Hartridge, H., and Hill, A. V.: The Transmission of Infra-Red Rays by the Media of the Eye and the Transmission of Radiant Energy by Crookes and Others Glasses, Proc. Roy. Soc., London, s. B **89**:56, 1915-1917.

5. Richardson, A. W.; Duane, T. D., and Hines, H. M.: Experimental Lenticular Opacities Produced by Microwave Irradiations, Arch. Phys. Med. **29**:765 (Dec.) 1948.

6. Daily, L.; Wakim, K. G.; Herrick, J. P., and Parkhill, E. M.: Effects of Microwave Diathermy on the Eye, Am. J. Physiol. **155**:432 (Dec.) 1948 P.

7. Salisbury, W. W.; Clark, J., and Hines, H. M.: Exposure to Microwaves, Electronics **22**:66 (May) 1949.

8. Cogan, D. G.: Lesions of the Eye from Radiant Energy, J. A. M. A. **142**:145 (Jan. 21) 1950.

damaging microwaves were about 12 cm. in wavelength and that the resulting opacities very likely were due to the thermal effect of the wave absorption. A support for this view is the well known thermogenic properties of microwaves, while evidence of any other mechanism of denaturization within the tissues is lacking at present.

After due consideration of the accumulated evidence it was thought to be of interest to study the effectiveness of 3 cm. pulsed microwave radiations in the production of ocular damage. Such investigation would serve to introduce a new segment of the microwave spectrum for observation and would aid in determining possible hazards with the use of radar type wave generators.

PROCEDURE

The studies were divided into two units: (1) the irradiation of intact rabbit eyes in vivo to determine any observable damage to the eye and (2) the irradiation of excised beef eyes to determine the temperature gradients produced by microwave energy on eye tissues.

Throughout the experiments on rabbits in vivo, a 3 cm. pulsed wave microwave generator was employed which delivered between 34 and 67 watts average power output as regulated. The pulse duration was 1 microsecond with a pulse rate of 1,000 per second. Care was exercised to avoid standing waves in the wave guide. The albino rabbits used in these studies were irradiated on a polystyrene pedestal which stool on a wood laboratory table. The animals weighed 3 to 5 Kg., and were anesthetized with pentobarbital sodium, 35 mg. per kilogram of body weight. Drops of atropine and tetracaine (pontocaine®) hydrochloride were applied to the eyes previous to exposure in order to minimize irritability and assure a uniform pupil size. The wave director was alined directly on and at a right angle to the optic axis, and the cornea was 5 cm. from the director cone in all cases.

These animals were maintained on sufficient water and standardized laboratory food during the experiments to insure an adequate diet. They were kept in well ventilated rooms moderately lighted with indirect sunlight. The eyes were inspected by use of a hand slit lamp and ophthalmoscope prior to irradiation, immediately after irradiation and at daily intervals.

The in vitro temperature studies were carried out using fresh beef eyes of standardized size and uniform initial temperature previous to exposure. The eyes were irradiated on a polystyrene pedestal and the temperatures recorded by means of thermocouples which were inserted just posterior to the cornea, at the anterior surface of the lens, at the posterior pole of the lens and just anterior to the retina. Temperatures were taken immediately after exposure. The thermocouples were of the iron-constantan type housed in hypodermic needles, as described by Tuttle and Janney.[9] Potentials were recorded on a Leeds and Northrup type 8662 potentiometer, and temperature measurements were calibrated with a Bureau of Standards thermometer. The excised beef eyes were irradiated by 3 cm. pulsed wave microwaves and by 8.5 and 12.25 cm. continuous wave microwaves in order to determine comparative thermal gradients induced by these various agents.

RESULTS

When 21 rabbits were irradiated by 3 cm. pulsed microwaves under the conditions delineated in the table, 16 animals were found to show opacities in the eye within 60 days. The right eyes were exposed in all cases, the left eyes serving as controls along with the preexposed right eyes. The first 12 animals received 67 average watts output for three to five minutes at 5 cm. distance. In this group there were nine corneal opacities, four opacities in the anterior segment of the lens and six cases of extraorbital burns on the eyelids or facial area superficially. Opaci-

9. Tuttle, W. W., and Janney, C. D.: The Construction, Calibration and Use of Thermocouples for Measuring Body Temperatures, Arch. Phys. Med. 29:416 (July) 1948.

ties of major size were accompanied with extraorbital damage, while minor opacities were not. No damage was observed in three animals.

A second group of nine animals was given exposures of smaller intensity over longer periods to observe whether under such conditions the opacities, if produced, would be found in the same areas of the eye or would be accompanied with extraorbital damage. Of the nine animals exposed to these energies, seven were found with corneal opacities, three were observed to have opacities in the anterior segment of the lens and four displayed evidence of extraorbital damage. No damage was observed in two animals of this group.

The character of the observable impairment to the tissues of the eyes varied from small opaque masses on the cortex of the anterior segment of the lens and

Orbital Damage Due to 3 Cm. Pulse Radiations at a Distance of 5 Cm.

	Energy Conditions					
	Average Watts	Minutes Irradiation	Average Watts × Minutes	Corneal Opacities	Anterior Lenticular Opacities	Extraorbital Irradiation Damage
First group						
1	67	5.0	335	+	−	−
2	67	5.0	335	+++	+	++
3	67	5.0	335	+++	+	++
4	67	4.0	268	++	−	+
5	67	4.0	268	++	+	−
6	67	4.0	268	−	−	−
7	67	3.5	235	+++	+	++
8	67	3.5	235	++	−	+
9	67	3.5	235	++	+	+
10	67	3.0	201	−	−	−
11	67	3.0	201	+	−	−
12	67	3.0	201	−	−	−
Second group						
1	60	14	780	+++	+	++
2	42	12	504	++	+	+
3	42	10	420	+++	+	+
4	42	10	420	+	−	+
5	34	18	612	−	−	−
6	34	18	612	−	−	−
7	34	15	510	+	−	−
8	34	25	850	++	−	−
9	34	20	680	++	−	−

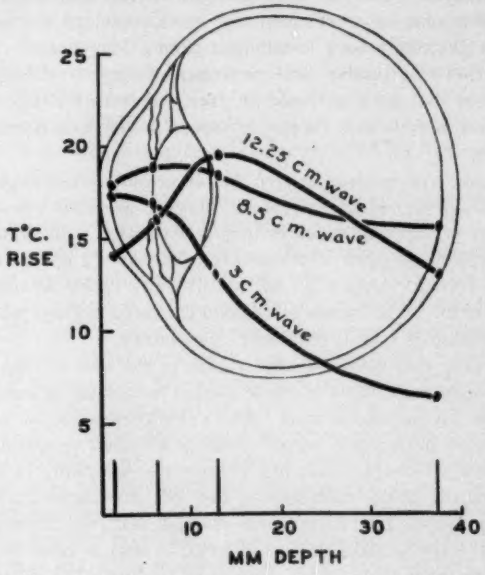
the inside surface of the cornea to complete opacifications of the cornea, which obscured any view of the interior of the eye with the slit lamp or ophthalmoscope. When corneal opacification presented such a problem, the lens was removed and examined by means of a slit lamp. A predominantly greater degree of damage was found in corneal tissues than in the anterior segment of the lens. The masses found in the anterior lens segment were situated between the pole and equator of the lens, typically represented as crescent or stellar masses partially circumscribing the pole.

Under the conditions described, no opacities were observed in the posterior segment of the lens. This was of considerable interest because previous workers [10] had reported posterior opacities to be induced by microwaves of longer wavelength. The impairment which was observed in the ocular tissues became evident from

10. Richardson and others.[5] Daily and others.[6] Salisbury and others.[7] Cogan.[8]

three to 60 days following the irradiations. Extraorbital burns, if found, were evident immediately following exposure to the field, in their first stage of development. These facial burns varied from 1 inch (2.5 cm.) in diameter to one third of the right facial area. In three cases the orbital damage was sufficiently great that the eye eventually deteriorated.

Since it had been demonstrated by previous workers [10] that exposure of the eye to high energy 12.25 cm. continuous microwaves caused opacities in the posterior portion of the lens, rather than in the positions described in these studies, it was considered of benefit to determine the gradient of induced temperature in the eye under each condition. In order to permit a better delineated evaluation, three conditions were selected for study—irradiation by 3 cm. pulsed microwaves, as well as by 8.5 and 12.25 cm. continuous microwaves.



Temperature gradients in beef eyes induced by microwaves of 12.25, 8.5 and 3 cm. wavelengths.

When 24 eyes of beef were irradiated under these conditions, eight in each group, three distinctly different gradients were induced in the eye tissues. The results may be seen in the figure. Preliminary experiments were carried out to equilibrate roughly the effective energy uptake as measured by the temperature induced by the three agents. In the controlled studies which followed this preliminary work it was found that, whereas the highest temperature induced by 12.25 cm. radiation was measured at the posterior pole of the lens, the highest temperature from 8.5 cm. radiation was at the anterior portion of the lens and the peak temperature induced by 3 cm. pulsed radiation occurred in the anterior chamber. The figure more specifically delineated the gradients. Since the 3 cm. studies include the two variables, wavelength and discontinuity of wave propagation, one is not

permitted an absolute statement regarding the effect of wavelength, but it was believed that the additional results of the 8.5 cm. microwave studies helped to support a view that the position of maximum absorption varies with wavelength when eye tissue is exposed to microwaves. A statistical analysis of the 12.25 and 3 cm. gradients showed a significant difference at the 1 per cent level of confidence at the posterior pole of the lens and a significant difference at the 2 per cent level of confidence inside the cornea.

COMMENT

It is apparent from these studies that damage to the eye is possible in 3 cm. microwave fields such as are used in "radar" work, if the energy intensity is of sufficient magnitude. However, since this pathological change is so closely accompanied with impairment to the more vascular superficial tissues containing normally adequate pain receptors, there is some question whether ocular damage is probable under most conditions. Nevertheless, one cannot exclude the possibility that a worker in close proximity to a low output over a long period or a high output over a short period may receive some permanent damage to the eye. Tests in our laboratory showed that the eyes could be protected from the high energy fields by covering the head or eyes with copper or bronze screen wire if cut to fit snugly to the skin surface.

In the production of opacities in vivo the power outputs are expressed in average watts because the effects of microwaves in biologic work are commonly associated with temperature production, which in turn is associated with average watts. The value of the microsecond pulse which occurs 1,000 times a second is approximately 1,000 times the average value, e. g., 6.7×10^4 watts (pulse wave) for 1.0×10^{-6} seconds pulsed at 1.0×10^3 times per second equals 67 average watts. In general, this type relationship is typical of "radar" generators.

While conceding that a precise explanation of the biologic mechanism contributing to the opacity formations in these studies would be of value, it was not a purpose of these investigations to do this. The correlation of temperature production with tissue impairment would point to elevated temperature as a major factor. This view, of course, does not exclude the possibility of concomitant and subsequent physicochemical mechanisms not yet investigated. Of considerable interest was the finding that extraorbital damage occurred immediately following exposure, whereas ocular damage was observable after a lapse of several days.

SUMMARY

A direct, single exposure of rabbit eyes to 3 cm. pulsed microwaves (radar type) at 5 cm. distance and 67 watts average output resulted in opacities in the eye after a delay of two to 60 days. These opacities were found to occur inside the cornea and on the anterior segment of the lens. Such ocular damage often was accompanied with damage to the superficial extraorbital tissues.

When induced temperature gradients in excised eyes of beef were plotted, the maximum temperature agreed well with the site of damage found in the in vivo irradiations.

It is suggested that reasonable precaution be used in the exposure of the eye to "radar" energy fields.

C. J. Imig and Barbara F. Randall assisted in this work.

A THEORY OF OCULAR DOMINANCE

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DOMINANCE in the broadest sense consists of any sort of physiological preeminence, priority or preferential activity of one member of any bilateral pair of structures in the body. Thus, to write right handed or left handed is to have a dominant hand. To start walking, or climbing or putting on one's trousers always with the same foot is to have a dominant foot. The discovery of dominance is lost in antiquity. Handedness has been known for so long that there are words in common speech, such as the French *gauche*, and the English "gawky," which allude to it. Eyedness, however, is supposed to have received its first scientific mention in 1861, by G. M. Humphrey.

In neurology, a first order expectation is that the lateralities (sidednesses) of the various dominances in a given individual will agree. This is because one primary laterality is in position to dictate the lateralities of most of those dominances which find expression in either sensory or motor ways. This basic sidedness is cerebral: The individual is either left brained or right brained, has either the left or the right cerebral hemisphere as the dominant or major hemisphere. If he is left brained, he has his centers for motor, auditory and visual speech, verbal auditory memory, and perhaps primary auditory sensation, in the left hemisphere. Their counterparts in the right hemisphere are unused and can be removed without demonstrable effects. Simple anatomic decussations strongly predispose the left-brained person to be right handed. His "general laterality" is expected to be dextral; that is, most or all of his various motor dominances "should" be of right members.[1] If, in any two pairs of organs, the right member in one pair and the left member in the other are dominant, "cross dominance" is said to exist. Cross dominance is always under suspicion in two ways: It is likely to cause difficulties of coordination, and it is likely to be the result of a secondary change of dominance forced on the individual.

In approaching the subject of ocular dominance, it is essential to strive to shed this attitude of expectation, this assumption that brainedness will be found to be the primary determinant. The problems of both sensory and motor dominance in vision are complicated—or potentially complicated—by the fact that the two eyeballs themselves do not constitute "the pair of structures," one member of which may

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1. The speech centers of a left-handed man may, however, be in either the right or the left hemisphere (Penfield, W. G., and Rasmussen, T.: The Cerebral Cortex of Man: A Clinical Study of Localization of Function, New York, The Macmillan Company, 1950).

be expected to be dominant in some way or in several ways. Superficially, the retina in each eyeball is a complete and unitary sense organ. But two portions of each retina are respectively connected separately with the two lateral halves of the brain. The group of oculorotatory muscles inserted on one globe turns that globe only. Only in that one respect is the group a lateral unit—the left medial rectus is yoked with the right lateral rectus in the conjugate components of binocular movements but pairs with the right medial rectus in the disjunctive components. Both complete sets of muscles are involved when either eye is stimulated to move, for neither eye ever moves alone, except in some laboratory situation. Both sets of muscles respond to innervation from either cerebral hemisphere—from either occipital oculomotor center or from either frontal voluntary eye movement center. Each labryinth and each cerebral hemisphere continuously send tonic innervation to the ipsilateral adductors and the contralateral abductors. Brainedness may affect the symmetry of these innervations (as in the blind); but if there is then an ocular deviation toward the side of the dominant hand, it is conjugate—not a deviation of one eye.

As regards any inevitable determination of eyedness by brainedness, "all rules are off," and we should be prepared to find whatever we do find, without surprise. When we observe a dominance expressing itself in the visual system, we may know the individual's brainedness, but this may or may not have anything to do with the matter; in any case, any of several other things may be concealed from us. It may appear that a preference (as in sighting a rifle) is genuine, or the preferred eye may be preferred only because its mate is inadequate or abnormal, or because the way guns are built enforces the choice. It may seem that the mere one-eyedness of the activity (as in looking through a small aperture) is all that requires the use of only one eye, whereas the really essential activities for which the dominance thus expressing itself was developed are those in which both eyes engage. (It is just as customary and desirable to have a dominant hand, in bimanual activities, as it is to adopt one hand or the other unhesitatingly for a one-handed task.) It may seem that the overt expression of the dominance, such as maintaining fixation within the convergence near point, is self contained and is the whole of the matter, whereas the essential reason for the phenomenon may be deep seated, and even difficult to discover. It may even be that the real basis of a dominance is sensory, although its obvious manifestations are motor, or an underlying motor asymmetry may have sensory or perceptional consequences.

Least noticeable—or, at any rate, least noticed—of all is the possibility that in a single pair of organs there may be several dominances at once: If the activities of the organs are numerous and complex, it may well be that one member of the pair is dominant in some activities while the other is the dominant member in other respects. This is sometimes true of the hands, and certainly it is often true of the eyes.

How can we expect dominance to be exhibited within the whole sensorimotor visual system, where a single function, such as "fusion," may have both sensory and motor aspects well nigh inextricable, and where, unlike the situation in the manual system, there is no neat divisibility into topographic halves connected in anything like isolation to the respective hemisphere? Various authorities have

grasped various appendages of this elephant and have laid down as many conceptions and definitions of ocular dominance. Duke-Elder [2] states (page 1056):

> When the vision in the two eyes is unequal from some pathological or refractive reason, or when strabismus exists, the better eye attains a position of marked supremacy, but when the two are approximately equal in visual acuity there may be little evidence of dominance.

Crider [3] considers "sighting behavior . . . the criterion for the dominant eye." Zoethout [4] defines dominance in terms of sighting but then goes on to refer to it as an ascendancy of one retina over the other. Jasper and Raney [5] are sure that "ocular dominance in monocular sighting is predominantly a motor function since it has been shown that the dominant eye . . . is *not* related to the eye possessing the best visual acuity." Washburn (Washburn, Faison and Scott [6]) defines dominance as "the right or left visual field tending to remain in consciousness longer than the other one" in retinal rivalry.

There is a great confusion of tongues here, and the different conceptions of dominance cannot possibly be reconciled with one another. Visual acuity determines dominance? Sighting indicates dominance but pays no attention to visual acuity. Sighting is therefore deemed motor, in a *non sequitur*. But, rivalry indicates dominance? Yet rivalry contains no motor elements. Nor could the only kind of rivalry considered by Washburn—color rivalry—be influenced by visual acuity! No wonder, perhaps, that the average refractionist who is seeking to determine a patient's dominant eye does so by asking the patient whether he is right handed or left handed, and supposes this method to be as proper as any.

Actually, a surprising number of big and little asymmetrical visual and ocular phenomena have been advanced as criteria of the sidedness of dominance. Many have been incorporated into tests, applicable in commonplace vision or with simple apparatus. Some of these tests are so widely used that they may be called standard. Yet even a glance at any long list of criteria should suggest at once that the tests do not all test for the same essential thing—that, in short, there must be two, or three, or perhaps a sizable number of different kinds of dominance manifested in the visual system. At the same time, one must not suppose for a moment that every way in which dominance expresses itself marks a different kind of dominance, any more than writing handedness and shaving handedness and coffee-stirring handedness are different kinds of handedness—they are all motor, which is the only kind of handedness there is (apart from inequality in the fineness of tactual discrimination by the separate hands, which is probably a result of one hand getting more practice).

I have compiled a rather long list of criteria of ocular dominance; but I stopped searching at the point of diminishing returns, and I would not claim the list to be really complete.

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2. Duke-Elder, W. S.: Text-Book of Ophthalmology, vol. 1, St. Louis, C. V. Mosby Company, 1938.

3. Crider, B.: A Battery of Tests for the Dominant Eye, J. Gen. Psychol. **31**:179-190, 1944.

4. Zoethout, W. D.: Physiological Optics, ed. 4, Chicago, The Professional Press, Inc., 1947.

5. Jasper, H. H., and Raney, E. T.: The Phi Test of Lateral Dominance, Am. J. Psychol. **49**:450-457, 1937.

6. Washburn, M. F.; Faison, C., and Scott, R.: A Comparison Between the Miles A-B-C Method and Retinal Rivalry as Tests of Ocular Dominance, Am. J. Psychol. **46**:633-636, 1934.

SOME CRITERIA OF DOMINANCE

It has sometime been considered that the dominant eye is:

I

1. The eye whose image in a rivalry situation is in consciousness for more of the time than that of the other eye.

2. The eye which alone sees movement in the Jasper-Raney ambiguous phi situation.

II

3. The eye with which one alines a finger or pencil in pointing at another object when both eyes are open.

4. The eye regularly chosen for sighting or looking when both eyes cannot be used.

5. The eye used for looking when the subject supposes he is looking binocularly but is not.

6. The eye with which the subject notices the lesser "jump" in a cover test.

7. The eye affording the better marksmanship (with rifle, pistol, etc.).

8. The eye whose occlusion (as for the elicitation of latent hyperphoria) leads to the greater uneasiness and the less self confidence in locomotion, etc.

9. The eye whose images of alined near and far objects do not appear to shift laterally when fixation alternates between the two objects, regardless of the eye with which they are alined.

III

10. The eye which is actually fixating when fixation disparity exists.

11. The eye in which no portion of any horizontal or vertical heterophoria resides.

12. The eye which in strabismus is neither deviant nor amblyopic.

13. The eye whose image is less readily ignored or suppressed, as in training in monocular microscopy, etc.

14. The eye which with bifixation at 20 feet deviates less if covered.

15. The eye which continues to fixate a target pushed up within the convergence near point.

16. The eye which diverges less, or not at all, when covered after a bifixation target has been brought and held 3 inches from the face.

17. The eye which having diverged under cover from a 3 inch bifixation target recovers its vergence when uncovered again, or recovers fixation faster than the other eye does.

IV

18. The eye before which one holds a card to read it.

19. The eye with which one looks more nearly perpendicular to a surface when asked to face that surface squarely.

20. The eye which is harder to close alone or to wink with.

V

21. The eye having the higher visual acuity.

22. The eye which in physiological diplopia of an object affords the more substantial-seeming image.

23. The eye which in physiological diplopia of a light affords the brighter image.

24. The eye whose after-image of a light persists longer.

25. The eye on the side of the dominant hand.

Group I Phenomena.—These, items 1 and 2 above, are asymmetries in which, so far as I can see, there is not the least possibility of an oculomotor element. In laterality, they could not be expected to agree with brainedness except by chance, for brainedness cannot conceivably dictate the sensory-perceptual behavior of one whole retina as compared with that of the other whole retina—"retina," of course, being used here in the physiological sense, to embrace the anatomic retina and all of the central nervous apparatus that mediates its sensory-perceptual role.

Rivalry asymmetry is cited in many lists of dominance "tests," although certainly no one ever bothers with it in clinical practice. There have apparently been few quantitative studies of it, but one such study was that by Washburn, Faison and Scott.[6] Their 58 subjects were each given, at two distances, what is probably the best of all "sighting dominance" tests—the Miles A-B-C test (Miles[7]). They were also made to experience haploscopic rivalry between areas of pure red and areas of pure blue. Record was kept of the time periods during which one color or the other stood alone in consciousness, and the "purple" time was halved and the halves arbitrarily added to the "red" and "blue" times. The per cent excess of total red or blue time was assumed to indicate "the amount by which one eye was dominant over the other," excesses of less than 10 per cent being regarded as insignificant, excesses of 10 to 20 per cent as indicating weak dominance and excesses of 20 per cent or more as indicating strong dominance.

Washburn's analysis of the results is involved and not wholly logical. In making my own analysis, I find that 33 of her subjects showed sighting dominance with strong or weak rivalry dominance of the same eye. Seven others had sighting dominance coupled with strong or weak rivalry dominance of the other eye. Seventeen others had sighting dominance but no rivalry dominance. One subject had no dominance of either kind. No subject was found to have rivalry dominance in the absence of sighting dominance.

Washburn assumed that only one kind of dominance was involved and that the rivalry test could not possibly give a false indication of its laterality. So, since she found that more of her right-handed subjects were called right eyed by the Miles test than by the rivalry test, she accused the Miles test of being biased by the handedness of the testee, whereas it is the only surviving sighting test which is entirely free from such bias.

Washburn thus completely missed the point of her own work, which clearly showed that sighting dominance and rivalry dominance are entirely independent kinds of ocular dominance, not merely different manifestations of the same under-

7. Miles, W. R.: Ocular Dominance Demonstrated by Unconscious Sighting, J. Exper. Psychol. **12**:113-126, 1929; Ocular Dominance in Human Adults, J. Gen. Psychol. **3**:428, 1930.

lying physiological asymmetry. Each of them may often "go with" hand dominance (or with brainedness), but it is then only coincidentally that they go with each other. I am unable to see why their lateralities should be expected to agree, for I cannot imagine a common physiological basis for them. Moreover, it needs to be pointed out that, whereas sighting laterality may be "switched" in ontogeny by a marked asymmetry of visual acuity for near or far, color rivalry should be immune to this, and the laterality exhibited by the individual should always be "essential" and never "imposed."

Whether the lateralities of color rivalry and form rivalry always agree or not seems never to have been determined. One could not say that disagreement here would be "expected," but neither would it be surprising. Proper analysis of sufficient data might show that color rivalry and form rivalry dominances are as distinctly different kinds of dominance as the rivalries are different kinds of rivalry.

The kind of nonmotor holocular (whole eye) dominance mentioned in item 2 is one which Jasper and Raney [6] discovered as a by-product of their ingenious study of a purely perceptual visual manifestation of brainedness laterality. The latter cerebrohemispheral visual dominance will have to be explained here if holocular phi dominance, in which we are more directly interested, is to be made clear.

"Phi" apparent movement is seen when spatially separated motionless lights are flashed successively or alternately with appropriate timing. The subject perceives a single light moving from one location to the other. Jasper and Raney placed two lights at 38 and 142 cm. in the sagittal plane of the subject, who kept his bifixation on the locus of one light or the other during the alternations of the two lights at a rate of 3 every two seconds. For either eye taken by itself, this situation predisposed to the perception of a single light moving from side to side in one half of the visual field or the other, depending on which stimulus was being fixated. The subject kept both eyes open, however, and a typical test went like this: The subject bifixated the far light. Whenever this was turned off and the near light was turned on, there was a basis for seeing simultaneously a light moving rightward (with the left eye) and the same light moving leftward (with the right eye). When the near light was turned off and the far one again came on, there was a basis for seeing the light to the right move leftward and the one to the left move rightward, the two coming together and becoming one light. After a few minutes' observation, the subject began to perceive movements. But the physical impossibility of one light splitting into two equal ones (or of one light moving in two directions at once) was rejected, and as the lights were alternated the subject saw as if he had one eye closed; that is, he saw either a light moving from side to side at the right of the field midline or one moving from side to side at the left of the field midline. Let us suppose that this particular subject saw movement on the right. This meant that material on the temporal half of the left retina was finding representation in consciousness, while that on the temporal half of the right retina was not. When the subject then, for a period, fixated the near light, he would again see a moving light only in the right half of the field. The material on the nasal half of the right retina was then being seen, with that on the nasal half of the left retina somehow suppressed.

In this performance the visual cortex of the left occipital lobe, receiving from the temporal half of the left retina and the nasal half of the right retina, was "dominant" over the right occipital cortex. In respect to the perception of apparent movement in this situation, the subject's left brainedness made him a diagrammatic hemianope, although his entire visual system was normal. Jasper and Raney obtained such responses from 34 of their 42 subjects, 30 of whom proved to be left brained and four right brained by this criterion. Their handednesses were in keeping, apparently without exception. But in the same subjects there were only

fortuitous relationships between cerebrohemispheral phi laterality and sighting laterality, and between the latter and handedness. The investigators thus confirmed the opinion that sighting dominance is not a good indicator of "general laterality," for it could not be argued here (as it might be with handedness) that any imaginable "social pressure" had falsified the apparent brainedness in some of the subjects by enforcing a switch of phi dominance from one side to the other.[8]

Seven of Jasper and Raney's subjects did not manifest this cerebrohemispheral dominance. These subjects saw, instead, a movement on the right when fixating one of the lights and a movement on the left when fixating the other. They showed a dominance for the perception of phi movement, but it was a holocular dominance, of one "retina" over the other, despite the division of each retina between the opposite sides of the brain. Six subjects saw the movements with the right eye only, the seventh being left eyed. Still one other subject in the experiment saw movements, but so inconsistently that his condition had to be called indeterminate.

It was probably only the particular character of Jasper and Raney's experiment that made it seem that each subject had either a cerebrohemispheral visual-perceptional dominance or a holocular visual-perceptional dominance. Each subject may have had both, but he could not exhibit both; so he showed the kind of dominance which was in some sense the stronger. This assumption erects a concept of dominance between dominances; but there is precedent of sorts for this, as when a left-eyed man is right handed but prefers to shoot a gun from the left shoulder, his eyedness being stronger than his handedness in that one situation at least.

It remains to be determined experimentally whether the lateralities of holocular phi dominance and holocular rivalry dominance for color and/or form agree. The possibility also remains that with haploscopic rivalry targets each of which was vertically bipartite, a subject fixating their midlines might experience something capable of being related to the cerebrohemispheral visual laterality which Jasper and Raney found.[8a]

At any rate, the phi and rivalry phenomena show clearly that there is at least one kind of "ocular dominance" that is not connected with brainedness and is also in no way related to whatever essential asymmetry underlies sighting dominance.

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8. Penfield and Rasmussen, referred to in footnote 1, offer much evidence that the traces of visual memory images are laid down in perfect duplicate in the cortex of both temporal lobes, even in a person with strong handedness, etc. These traces are not "hemianopic," for electrical stimulation in one lobe can set off a hallucination that occupies the whole visual field.

8a. The underlying basis of their particular phenomenon can probably be made to express itself in situations other than the "phi" one. A patient has been described (Bender, M. B., and Furlow, L. T.: Phenomenon of Visual Extinction in Homonymous Fields and Psychologic Principles Involved, Arch. Neurol. & Psychiat. **53**:29-33 [Jan.] 1945) who, while recovering from a blow on the left occipitoparietal region, could see a stimulus offered in the right half of his visual field only when there was no stimulus present in the left half. The authors tried to relate this phenomenon to the mechanism of rivalry. It seems possible that a right cerebrohemispheral dominance was created (or exaggerated) by the injury, with the visually dominant (uninjured) hemisphere exercising its domination only when it was "working," visually. Perhaps the Jasper-Raney phenomenon can be described in just about the same words. It would be difficult to devise an experiment which would show how much domination is actually being exerted when the dominant and nondominant moieties of the system are not being pitted against each other by symmetrical stimulation. A fundamental question is: Is a structure in itself dominant, or is it only the activity of the structure that can be said to be dominant?

Group II Phenomena.—It is in these phenomena that one readily recognizes the familiar laterality expressed by such terms as "dominant eye," "master eye," "sighting eye," "preferred eye" and "directing eye in binocular vision." Javal gave it the last name, and it is incomparably the best of these terms, though the least used. It does not allow the user to forget that dominance is a phenomenon of binocular vision: An eye does not become dominant only when the other eye is out of action.

There is what one might call "a mysterious something motor" about all the phenomena of group II. But it is not anything so evident to introspection as a kinesthetic feeling of steadier, or stronger or quicker-acting mobility of one eyeball. One consciously or unconsciously chooses a particular eye to sight with, but not for anything like the reasons for which one chooses a particular hand to throw a stone with. To my mind, it is this very elusiveness of just what it is that is motor about "sighting dominance" that has kept its motor nature from being unanimously agreed on thus leaving room for such confusions as Washburn's, and has consequently kept its basis from being elucidated.

Whether the best name for it is "sighting dominance" or something else, there is certainly a kind of ocular dominance that is utterly distinct from rivalry or phi dominance, or any kind of purely sensory-perceptional visual laterality that may remain to be discovered. It is motor in its general character. But do the group II phenomena constitute this motor dominance in and of themselves? Or are they only some of the consequences of an asymmetry which in its true nature is perhaps much more important and "interesting" (because unexpected!) than any of them? Temporarily, I shall seem arbitrary when I now assert that the group II phenomena are one-eyed expressions of an asymmetrical but binocular phenomenon, for which I propose the term "directional dominance." "Sighting dominance," then, is not a kind of dominance, but is only one of the ways in which the laterality of directional dominance reveals itself in monocular vision.

The phenomena assembled in group II were placed there because they suggest that the motor-dominant eye is "dominant" because, and in that, it is the eye with which one ascertains the direction in which a point lies with reference to the self as the center of subjective visual space, whether one is seeing binocularly or monocularly with either eye. We cannot, of course, go into the meaning and basis of this statement until we have examined—farther on—the whole mechanism of egocentric visual directionalization.

Group III Phenomena.—These phenomena are not really separable from those of group II, but are perhaps a little more easily seen to be secondary consequences of a dependence on one eye for the establishment of direction. The eye "non-dominant" in this regard has so little responsibility that no harm is done if it deviates from the point of regard, for, according to my hypothesis, the direction in which that point is seen to lie is wholly determined by the dominant eye, in any one who has one. The system as a whole is indifferent to the motor behavior of the non-dominant eye, just so long as it does not wander so far as to give rise to diplopia. It may wander as far as it likes if it is covered, or if it is uncovered but fusion is not being attempted, or if it is amblyopic or if it is suppressed. Not so the dominant eye: this eye must maintain steady aim even under cover, or an object continuously imaged on the other fovea will appear to be changing its direction when it is not.

Group IV Phenomena.—In the three respects enumerated here, the dominance of the directionalizing eye asserts itself in the sense that it is as if the eye were saying: "What's the use of being 'dominant' unless one has some privileges?" In the first two respects the eye is a "master" eye—it behaves as if it were lazy, as if it were disinclined to leave the primary position and were trying to insist that the "servant" eye do all of the vergences necessary to secure and maintain haplopia. This behavior would seem to be quite in keeping with the idea, offered here, that every movement of the dominant eye is fraught with spatial-perceptual significance. The movements of the other eye need only be those which will bring its image of the fixation point to the middle of its fovea. It does not matter what gymnastics it has to go through to accomplish this, for they do not affect its owner's orientation in his environment.

Group V Phenomena.—These, in my opinion, are probably all meaningless. Let us consider them serially:

The contention that the sighting-dominant eye is usually or always the subject's better eye, i. e., his more sharply seeing eye, has been disproved again and again, but it will not die out of the textbook literature. When a young refractionist is told never to prescribe so that the visual acuity of a nondominant eye will come above that of the dominant eye, he may not recognize the advice as bad; but he is likely to think it unnecessary, since the books tell him that the dominant eye automatically has much higher acuity, and is dominant for that very reason. As an antidote to this antiquated teaching, such studies as that of Gahagan [9] are valuable:

Gahagan examined 100 emmetropic university students for sighting laterality; 79 were right eyed and 21 left eyed. Forty had the same acuity (Snellen) with either eye. At least these, then, had an essential dominance, not one forced on them by a decidedly better eye. There were 27 others, with unequal acuities, but with the dominant eye having the less acute vision. Their sighting lateralities must also have been essential, raising the incidence of essential dominance to at least 67 per cent. If the "crossed" combinations in these 27 subjects were chance combinations, one might expect that there would have been another 27 with unequal acuities and with the dominant eye the eye with the more acute vision. Actually, there were 33 such subjects, six of whom [10] might then have been subjects in whom the laterality of dominance had been imposed by the laterality of acuity. But only in half of these—3 per cent of the "population"—would the enforcement have had to overrule an opposing essential tendency. It can thus be made to seem that only an extraordinary superiority in visual acuity is likely to determine which eye shall be the one preferred for sighting. Certainly, if one wishes to argue (from Gahagan's figures) that greater efficiency somehow inheres in having the better-resolving eye develop into a motor dominant one, it is disconcerting to find only 33 per cent of people enjoying this situation and almost as many (27 per cent) having, instead, the "worst possible" situation.

Items 22, 23 and 24 are under deep suspicion. I cannot confirm any of them in my own person, although I have a strongly dominant eye. There seem to be no

9. Gahagan, L.: Visual Dominance-Acuity Relationships, J. Gen. Psychol. **9**:455-459, 1933.

10. Assuming that the difference between 27 and 33 is significant, which, of course, it is not. The simple analysis offered here is my own, not Gahagan's. A statistician would criticize it as being overgenerous to the "better eye" theory of motor ocular dominance.

documented accounts of any quantitative validations of these three criteria. Indeed, as "tests" they are generally criticized because of their subjectivity. The character of all of them is exactly what one would expect in cases of pseudodominance in which the laterality is forced by a considerable difference in visual acuity due to uncorrected anisometropia, or a difference in retinal quality due to a mild pathologic condition in one eye. Even in the absence of such organic asymmetries, careless testing may make one or all of these tests seem to work. For example, if a distant point is fixated and a near object not exactly in the sagittal plane is seen in physiological diplopia, one of its images will be more nearly foveal than the other and will naturally seem more "corporeal" or more vivid, without dominance being in question at all. A weak light seen in physiological diplopia may have one of its images near or in a fovea, and will then look very different in brightness to the two eyes, regardless of any considerations of dominance. In fact, when I see in diplopia a spot of light in a dark room, I readily find that I am usually fixating it with my dominant eye, for if I let the two images slip slowly back together only one of them "moves" to combine with the other.

Of course, someone should take the trouble to determine experimentally whether these three criteria do not perhaps exist for some subjects, with a genuine basis in a true dominance. If they are ever real, they may represent a perceptual dominance to lay alongside rivalry and phi dominance. Or, if the depressed performance of the nondominant eye is a part-way stage in the development of a suppression, these criteria might even prove to be manifestations of directional dominance, like item 13.

Item 25 is included in the list only for its historical interest, if any. Otherwise, it is so ridiculous as to be beneath notice. I myself am strongly left eyed—and as strongly right handed. So is every fourth man I meet on the street. One man in three is right eyed and left handed (Duke-Elder [2]).

THE TWO FACTORS IN EGOCENTRIC DIRECTION

In the visual perception of space, localization is all-important. The absolute and relative positions of points with respect to the self, and changes of position, i. e., movements, comprise all there is to perceive. Visual space itself vanishes if it is not kept in existence by being occupied and bounded by visual objects in particular positions, with all their parts in particular positions.

Two elements make up the position or locality of a visual point or object: its direction and its distance. We shall not be concerned here with the basis of the perception of distance, but we do have to consider the basis of egocentric direction.

Let us deal first with the monocular situation, without regard at first to eye movements. We may pretend for the moment that there are no eye movements, that there have never been any eye muscles. If, now, a visual stimulus (say, a star) is imaged at the center of the fovea, the cone visual cell most affected gives rise to nerve impulses in a particular optic nerve fiber. This fiber synapses in the lateral geniculate nucleus with a particular optic radiation fiber, which travels to the primary visual cortex (area 17) of the occipital lobe and terminates there on a particular ganglion cell. This cell being aroused, a star is seen, and is perceived as lying "straight ahead" from the eye. The activity of the particular foveal cone is the indicator of the real spatial position of the star and is what is generally

called a "retinal local sign." This "sign," however, gives no cue whatever to distance. Again, the star is not seen straight ahead "because that is where it is" or "because" it is imaged on a particular retinal receptor, but really because a particular cell in area 17 is brought into action. It is the activation of that cell that gives the subject his "sign" of the spatiality of the star. It is, then, the *cortical directional sign* of the star, and there is actually no such thing as a "retinal local sign." It is not the retina, but the cortex, that "sees" the star; and it is the particular spot in the cortex that sees, as lying in a particular direction, anything recorded neurophysiologically at that spot.

Suppose, now, that a second star appears, say, 10 degrees to the right of the first. Its direction relative to the direction of the first star, i.e., relative to the direction of the visual axis and the fixation point, is given by the perceptional properties of a cerebral cell which, so to say, lies 10 cortical degrees cortically rightward from the foveal-central cortical element. Area 17, although topographically banjo shaped, is isomorphic with an entire half-retina, which is to say that it is a map of the retina in the same sense that a distorted Mercator projection (although topographically a plane rectangle) is a map of the spherical surface of the earth's globe. Each locus in area 17 always does its seeing by way of a particular locus in the retina, as though it looked out through a tiny window in the retina at that locus, or as though the cortical element were, itself, in the retina. So, from every locus, every point, in the retina a line can be drawn through the perspective center of the eyeball (at the center of the entrance pupil) and on out into physical space. Each such line has, or is, a direction along which some particular fixed element in area 17 fixedly sees. Each such line is a direction with respect to the eye itself as a center—an oculocentric direction. The totality of oculocentric directions forms a cone, the axis of which is the direction line of the fixating point in the fovea. This is to say that any oculocentric direction is a direction with respect to the visual axis as a polar "zero."

Now we can begin to involve binocular considerations (with eye movements still left out of consideration). Let us select a point in area 17 in the left occipital lobe, which receives from a point in the temporal fundus of the left retina and gives the oculocentric direction "10 degrees directly rightward from the fixation point." To this same cortical point there is also connected a point in the nasal fundus of the right retina, whose stimulation also connotes a "star" 10 degrees rightward from the fixation point of that eye. It is because these two points in separate retinas "project" to the same cortical point, and therefore mediate homologous oculocentric directions, that they are "corresponding points." I do not know of any other really proper way in which to define corresponding points in normal vision.

This phenomenon of correspondence is due to the fact that each point in area 17 acts as a sensory fusion point and is one of the consequences, though not the *raison d'être,* of the partial decussation of the mammalian optic nerves. The nasal half of each retina and the temporal half of the other operate as if their cortical representations were locked in superposition or congruence, physiologically as well as anatomically. It is exactly as if two television cameras were connected to one receiver, so that the screen of the latter always contained a double exposure. Separate images of similar objects, or of the same object, could merge on the screen and become "single" images only if the objects were properly positioned on the

pick-up surfaces of the respective cameras. Just so it is with the conditions for diplopia and haplopia in binocular vision: Similar images must be positioned on sets of corresponding retinal points if they are to appear "single."

We can carry the analogy to television one valuable step farther. Suppose that the two cameras are aimed not at the same object or at two identical objects, but at entirely dissimilar objects. These cannot be seen merged and unified, but inevitably they must be seen superimposed on the screen. They must therefore be seen in a common direction by the observer, no matter where he is standing with respect to the screen when he looks at it. Similarly, in binocular vision, if different objects are imaged on corresponding retinal points, they are recorded at a single cortical locus and must be seen superimposed in some one direction. They are seen in diplopia, but the diplopia is homotopic.

This consideration leads to a paradox, which I shall formulate in the guise of a puzzle: Suppose my eyeballs could be hauled forth somewhat from the orbits without breaking any essential connections—far enough, say, that they could be rotated a bit and placed back to back. No object in the field of view of one eye could now be seen by the other. Suppose that a lamp which is objectively "at my left" happens to be imaged on my left fovea, and that a book which is physically "at my right" is imaged on my right fovea. The rules of the visual system dictate that I must see the lamp on the book and must see both objects in one direction. The puzzle is: What direction shall this be? The actual direction of one of the objects? But the other object has an exactly equal claim! The direction of neither? Then, why? And if in neither, then in what other direction—and why that direction?

What we now understand to be the basis of oculocentric direction cannot alone enable us to solve the puzzle. Something more, something else, takes hold of each cone of oculocentric directions and orients it, imparts to it a direction from the self. In the construction of the egocentric direction of a visual object, oculocentric direction is an indispensable element, but it cannot be the only one.

The second element is related to the eye movement system. So, to the hypothetical motionless eyeballs we have been discussing, we must now supply their full complement of rotatory muscles and their central nervous controls. We find that now, when an eye turns in the orbit under muscular action, an object imaged on the fovea (or an after-image developed there, which is more convenient to arrange for) is not seen permanently straight ahead, but is seen in the direction—any direction—in which the fovea is aimed at the moment. The eye muscles swing the entire cone of oculocentric directions about in an egocentric space. It is as if the muscles were attached to a frame around the oculocentric field, instead of (or as well as) to the eyeball. To return for a moment to our television analogy: It is as if whenever the camera was moved, some agency also swung the receiver about so that the observer looked at it in the same direction as the aim of the camera. Since he sees the televised material from the point of view of the camera (without regard to the location of the receiver), it is now as if he were stationed inside the camera and turned with it.

Something about the activities of the eye muscles is being continuously recorded, as if noted down by a stenographer in the association cortex. A record is being kept, and a continuous transcript of this record is telautographed to the perceptual "center" in which perceived egocentric directions are formed. Men's ideas have

changed, with time, as regards what it is that is thus recorded. It is no longer believed to be a stream of kinesthetic information from proprioceptors in the eye muscles. There are such proprioceptors, but they send nothing above the level of the midbrain. The record is one of the whole pattern of nerve messages that are being sent to the muscles. Modern psychology rejects the notion of a "sense of innervation," which, among other terms that have had some employment, was Helmholtz' expression for an unconscious awareness of the pattern of innervation. For want of any less clumsy term, I shall speak here of the record of innervation, or the innervation record factor, as the thing that gives to every oculocentric direction line an egocentric orientation.

HYPOTHESIS OF DIRECTIONAL DOMINANCE

An object seen haplopically—in "binocular single vision"—may carelessly be said to be seen "in the same direction with each eye" or "in a single direction with both eyes at once." But such phraseology would not be accepted by advanced students. A point cannot have the same direction for each eye, or from both eyes, for the two eyes and the point are not in one straight line. If, in binocular vision, a perceptually single point has a perceptually single direction, then this is a single direction from some single entity. The entity in question is the binocular visual self—for short, the visual ego.

If a point lies in homologous oculocentric directions from the respective eyes, it is seen singly. If seen singly, it lies in a single egocentric direction. Now, the point locus of the ego might be at any of many places; but any successful "sighting dominance" test shows it to be at the perspective center of one of the eyeballs. Why, now, should egocentric directions stem from one eye, even in binocular vision? One answer is that they could not stem from more than one point in any case. But why a particular eye, and not the other eye? Clearly, because an important element of binocular egocentric directionalization pertains only to that one eye. The haplopic object has, however, an oculocentric direction from each eye, and these homologous oculocentric directions are entirely on a par. It must, then, be in respect to the innervation record factor that the "dominant" eye is unique.

My thesis is that in any individual who has a motor-dominant eye an innervation record is kept only for the muscles of that eye, that this is what motor dominance really means and that this explains all the properties of the "dominant" eye—both in binocular vision and so far as they differ from those of the other eye when each eye is used alone.

The motor coordination of the eyes, as we know, is insured by a complex of synkineses and reflexes which operate to secure and maintain "fusion." The imaging of the same object on both foveas is brought about automatically. The binocular seeing of the object as single, and as lying in a single (egocentric) direction, follows. Whatever the innervation pattern going to one set of eye muscles may be, it is complemented by a particular pattern going simultaneously to the other set. There should be no need of keeping and using, for localizing purposes, two records of innervation when one would do as well as the two. In statistical work, if two coefficients always add up to unity, one does not bother to write down both of them. If one of them is on file, one always knows what the other must be.

The visual system sensibly avoids a source of confusion to itself by watching only one essential telautograph, instead of trying to watch two at once. In other words, the system becomes secondarily asymmetrical, and formerly one could only describe what happens by saying that a "dominant eye" has been adopted or developed.

Under this hypothesis, the single direction, in which the material imaged on a correspondent pair of retinal points is seen, is an egocentric direction into which the oculocentric direction of the point in the "dominant" eye is swung by the effect of the innervation pattern that happens to be traveling to the muscles of that eye. The innervation factor does not have to swing both the homologous oculocentric directions of the respective eyes, for the two cones of oculocentric directions are kept permanently congruent by a separate phenomenon—the fixed anatomic relationship of each area 17 to both ipsilateral half-retinas. If I will my eyes to turn up, and suppose them to have turned up, then any one thing or two things imaged on my two foveas at the time will be seen "up." Even if (as in my puzzle above) the eyes are statically back to back, so long as innervations can be started toward eye muscles, the lamp will be seen on the book and both will be seen overhead. Whether the voluntary innervations telling the eyes to turn up actually reach and actuate the muscles is of no consequence. It would be equally inconsequential whether any innervation even starts for the muscles of the nondominant eye, or not.

THE EVIDENCE

The hypothesis of directional dominance states that the essence of motor ocular dominance is the employment of the record of the innervations to the muscles of one eye only for the construction of binocular percepts of visual direction. If the hypothesis is correct, it should be found that movements of this one eye will cause changes in the egocentric directions of objects whose oculocentric directions are constant, whereas movements of the other ("nondominant") eye will cause no such changes. The immediate result of a change, or a changing, of the egocentric direction of an object is a translatory movement of the object across subjective space. The most direct proof of the hypothesis, then, would lie in any situation in which movements of one eye caused apparent movements of objects and similar movements of the other eye caused no such apparent movements of similar objects.

Such a situation is involved in item 9, above, and in one form or another was known to Hering and to Tscherning. Unaware of that, I (re)discovered one version of it some years ago and offered an explanation (Walls [11]). But at the time my ignorance of human vision prevented my understanding its full import.

The essential phenomenon can be displayed in a variety of ways. Perhaps the simplest is the one diagrammed in figures 1 and 2. In figure 1 *a*, near and far points *P* and *A* are alined on the visual axis of the dominant eye (with *P* shown in the sagittal plane and *A* close to *P*, for pictorial convenience). A screen conceals both *P* and *A* from the nondominant eye, but this eye is open. The observer "attends to" *A*. For the sake of the experiment, he can readily pretend that he is bifixating it. Unless he is one of those persons who have "utrocular" vision, he

11. Walls, G. L.: The Vertebrate Eye and Its Adaptive Radiation, Bulletin 19, Bloomfield Hills, Mich., Cranbrook Institute of Science, 1942, p. 90.

actually has no physiological basis for knowing that he is seeing A only monocularly, or for knowing with which eye he is seeing it. With fixation on A, the egocentric direction of both P and A is the line f-P-A. Now (fig. 1 *b*) the observer changes his "bifixation" to P. His nondominant visual axis (assuming adequate accommodative convergence) turns from an aim toward the unseen A to pass now through the unseen P. Neither P nor A appears to move as the nonseeing eye thus turns, and the egocentric direction of P and A remains the line f-P-A.

This result was, perhaps, "only to be expected." For why should movement of a covered eye influence the localization of things it cannot see? But, now, let us place the screen before the dominant eye and make the arrangement otherwise a mirror image of the previous one (fig. 2). Again, let "bifixation" be at first on A (fig. 2 *a*). The observer will now find that the egocentric direction of P and A is not the line f'-P-A, as might be expected, but the line f-P_R-A. For he

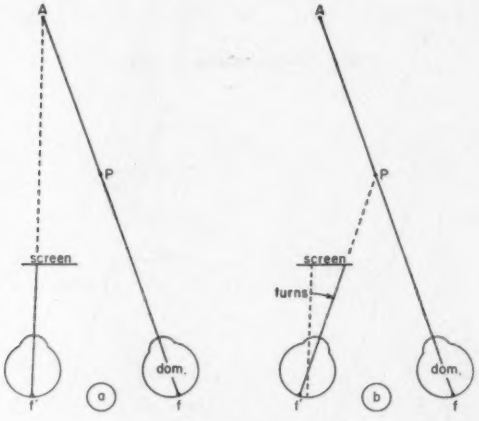

Fig. 1.—Explanation in text.

sees P and A as though the dominant eye could see them, seeing A as a fictitious right-eyed A (though seeing it where A really is), and P as though it were at P_R. This time, when "bifixation" is switched to P (fig. 2 *b*), it is only the nonseeing, "dominant" eye that turns—but the points P and A "move." Despite the fact that the eye seeing them is motionless, and despite the fact that their images on its retina are motionless, P and A both subjectively shift to the left, so that their egocentric direction, the line f-P-A_R, will still be the produced visual axis of the dominant eye.

The directionalization of a visual point seen by either eye alone or by both is, then, determined by the orbital posture of one directionally dominating eye, whether this eye sees the given point or not. This can mean only that the motor activities of the other eye are not made use of for the purposes of space perception,[11a] in accordance with the hypothesis.

(See footnote on next page)

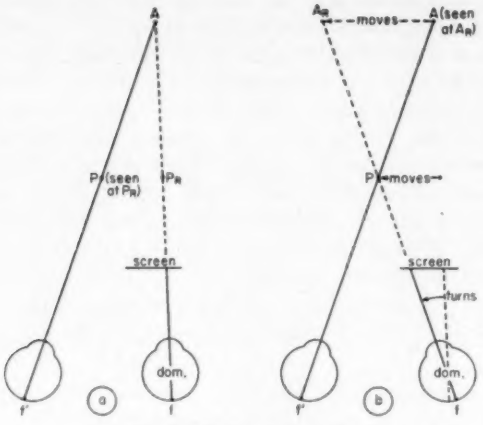

Fig. 2.—Explanation in text.

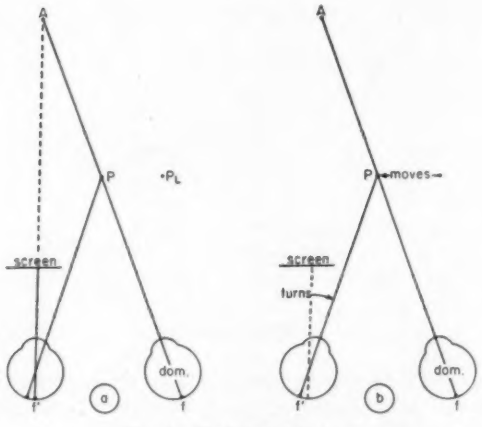

Fig. 3.—Explanation in text.

11a. This is not a typographical slip. I do mean to imply that any employment of the innervation record in the establishment of egoriginal distance (as well as direction) pertains to one eye only. Convergence is a valuable, though unreliable, cue to the absolute distance of a single object. It may be objected that my hypothesis implies that convergence could not cue distance unless the object were in the sagittal plane, so that the convergent innervation to the dominant eye would bear a regular relationship to distance. I would point out that all the evidence indicates that disjunctive innervations to the two medial rectus muscles are always symmetrical even when the convergence itself is asymmetrical, they being sent from the nucleus of Perlia, independent of asymmetrical innervation of the muscles for the conjugate component of the eye movement taking place. It is as if each medial rectus, in its excitation (and each lateral rectus, in its reciprocal inhibition) were two separate muscles: one actuated from the nucleus of Perlia and the other actuated from the bridge of the third nerve nucleus (for lateral deviations).

Essentially the same situation can be made a little more complicated, and somewhat more informative, by moving the screen so that P is visible to both eyes (figs. 3 and 4). The observer again "bifixates" A with the screen at first before the nondominant eye (fig. 3*a*). The point P as seen by the dominant eye (at P) is alined with A in the egocentric direction *f*-P-A; but there is now an extra P, P_L, off to the right. This is seen by the peripheral retina of the left eye, but is not seen as if at P. As bifixation changes to P (fig. 3*b*), the extra P slides into mergence with the P of the right eye to create a haplopic P. There is nothing contradictory to the hypothesis in this perception of a movement of one P due to a turning of the nondominant eye, for the image seen as moving is actually moving over the retina of the nondominant eye. Its oculocentric direction is changing, so of course the egocentric direction imparted to that oculocentric direction (by the posture of the dominant eye!) must change—for this reason alone, and not because

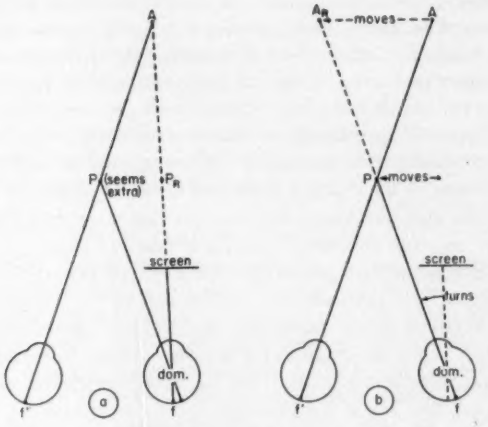

Fig. 4.—Explanation in text.

of anything the muscles of the nondominant eye or their central nervous controls are doing. Since A does not "move" and the P alined with it does not "move," there is no change in the egocentric direction of the line *f*-P-A.

Let the screen now be placed, instead, before the dominant eye, again permitting binocular fixation of P, but with A initially "bifixated" (fig. 4*a*). The observer will now find that the foveal P appears to be at P_R, so that the egocentric direction of P and A is the line *f*-P_R-A; and an extra P now appears off to the left. But when the dominant eye turns for the bifixation of P (fig. 4*b*), the diplopic images of P approximate and combine, and the now haplopic P takes the new egocentric direction *f*-P, while the A which the turning eye cannot see "moves" leftward, as it must seem to do, to remain perceptually alined with P in the new egocentric direction *f*-P-A_R.

The important consideration here is not the "complete" movement of A to retain perceptual alinement with P in this particular experiment, but the fact (thus demonstrated) that any movement of the directionalizing eye causes an

apparent movement of things concealed from that eye but visible to the other. It is this that explains why so many of the "criteria of dominance" are means of identifying the dominant eye. If, now, the whole list of phenomena in groups II, III and IV are reviewed, they can all be seen to be evidence for the hypothesis of directional dominance, in that they are explained by it and thus help to convert it into a proper theory of the nature of motor ocular dominance:

One sights or points with one eye because the visual ego is as though located in that eye, for the reason that egocentric direction lines initiate from the eye whose movements establish them. One consciously chooses this same eye for monocular tasks if one has had sufficient monocular experience to notice that localization is surer and steadier by way of the directionalizing eye. For the latter reason, a rifleman makes better scores with his sighting eye than with the other, for the motor wobblings of the disused eye do not then cause apparent wobblings of the gunsights and the target. One's unwitting dependence on one eye for the directional aspects of orientation comes to one's notice when an occluder has to be worn, for it is then soon found that one eye can be more comfortably spared (i. e., covered) than the other. Quick covering and uncovering of the directionalizing eye does not cause things to jump, since their egocentric directions are given by that eye and do not change if that eye moves (since the recorded change of innervation "cancels" the change of oculocentric direction). But uncovering a nondominant eye, which has deviated under the cover, suddenly introduces a diplopic image, and the extent of the diplopia is the extent of the jump.

Many a person does not know which is his dominant eye, but this does not necessarily mean that the dominance in such a case is "weak." The beginner at the monocular microscope may go through a rather long period of experimentation before settling down to a particular eye. This will be his dominant one. It has not been made dominant by the monocular practice, but has only been thus identified to its owner. After habituation, the microscopist becomes able to keep both eyes open without noticing the material on the table beneath the spare eye. But if he then tries to use that eye at the instrument, the contents of the field of view of the dominant eye are seen superimposed on the instrumental field, cannot be ignored and seem to interfere with good observation of the slide. For the nondominant eye to be the eye easier to "suppress" in such situations as microscopy and riflery seems logical, since to suppress the eye depended on for directions would impair orientation.[12]

Fixation disparity is a "plus or minus tolerance" that allows for the limitations on the ability of the binocular motor coordinating mechanisms to take and hold a setting of the eye muscles with anything like the precision they might have if they were made of metal, instead of living tissues. If, however, the triangular

12. The relative ease of closing the nondominant eye (item 20; Crider, B.: Eye-Closure Facility and Eye Dominance, J. Genet. Psychol. **58**:425-426, 1941) is closely related to this matter. A person who sets out to form a monocular habit, such as microscopy or shooting, at first closes the "spare" eye, which thus becomes easier for him to close than the used (dominant) eye. As he later learns to "suppress" the spare eye, he learns to keep both eyes open. But he will retain the greater facility of closing or winking the nondominant eye. Of course, a person may have a dominant eye and yet find it no easier to close one eye than the other if he has never had occasion to develop any monocular habits.

relationship of the two eyeballs and the point of regard is doomed to be thus inaccurate, it seems logical to relegate all of the inaccuracy to the eye whose variations of posture cannot induce corresponding falsifications of the perceived positions of objects.[12a] If, in the nature of living things, there must be some flutter of the spatial relationship of the two eyeballs to each other within the head, then it is only "common sense" to let one eye stay still so that all of the imprecision is imparted to the other—particularly if minor deviations of that eye can do no harm. If direction is perceived via only one eye, and some feedback system steadies the aim of that eye in obedience to the direction registered, then, obviously, there could be no such system for the other eye. Only the fixation and fusion reflexes serve to keep the nondominant eye aimed approximately at a target, while something more may control the aim of the dominant eye, which should be correspondingly more perfect.

The major monocular deviations which express themselves in tests of heterophoria and in frank strabismus are, again, things which are permitted to the nondominant eye but forbidden to the dominant, by the essential nature of dominance as a reliance on one eye for the perception of direction. The motor conservatism of the dominant eye, which looks like "laziness" in the oculorotatory phenomena of group IV, is only to be expected if every movement of that eye is pregnant with significance for space perception; the other eye, whose movements mean less to the organism, is the logical one to which to assign those vergences which have for their only value the maintenance of haplopia.

A FURTHER POSSIBILITY

In their analysis, Jasper and Raney were able to see only "three possibilities of asymmetric function in the visual mechanism: 1) dominance in the motor control of the eyes; 2) dominance in the receptor mechanism of the eye itself; and 3) dominance in the central projection areas of the two eyes." Their possibility *2* probably does not exist, for both the kinds of phi dominance they found, and rivalry dominance as well, are subsumed by their possibility *3*. They decided that sighting dominance is "predominantly a motor function" for the sole reason that it is unrelated to the laterality of asymmetrical visual acuity. In their minds, sighting dominance became "motor" by default, and not because they could see in just what way it was "motor." Not being much interested in it, they were at no pains to demonstrate how sighting dominance could possibly exemplify their possibility *1*, as a dominance in the motor control of the eyes.

My theory, as set forth above, embodies a possibility not entertained by Jasper and Raney, namely, an asymmetrical employment of symmetrical acts of motor control, secondarily, as determinants of perceptual directions. It does not assert that there is anything one sided about the fluxes of innervation, themselves, going to the left-eyed and right-eyed sets of oculorotatory muscles. Nevertheless, there does seem to be a possibility of just such an asymmetry, which would rescue Jasper

12a. Ogle (Ogle, K. N.: Researches in Binocular Vision, Philadelphia, W. B. Saunders Company, 1950, pp. 89-93) describes ingenious experiments by which he was able to find that in his own binocular vision all of the fixation disparity related to the nondominant eye. This has been assumed for about as long as fixation disparity (formerly called "retinal slip") has been known to exist; but experimental proof has been lacking.

and Raney's first possibility from discard. I do not, however, advance it as a necessary portion of my theory of directional dominance, nor do I (at this time) consider it any stronger than just a possibility.

The idea might be called "the steering gear hypothesis of eye movement." The older reader will recall that formerly the steering gear of an automobile involved a steering arm which was attached to one front wheel and was turned, through a split nut and worm, by the rotation of the steering wheel. The steering wheel turned only this one front wheel directly; and it was an independent tie rod, running from one front wheel to the other, that turned the other road wheel—the right one on American cars and the left one on British ones.

In all voluntary eye movements the system may operate in much the same way. All voluntary innervation of the eye muscles perhaps goes to the muscles of one eye only, just as the "voluntary" steering wheel in my analogy turns one wheel only. It seems a little foolish to suppose that in a squinter with an amblyopic eye any voluntary innervation ever goes to that amblyopic eye, for it could not be specifically aimed anywhere without a conscious allowance being made for the angle of squint. There may be no more need for voluntary innervation of the nondominant eye in a normal visual system. The "tie rod" would be furnished by postural reflexes and synkinetic accommodative convergence in an amblyopic (or an amaurotic) person, and by these factors and the "psycho-optic" fixational and fusional reflexes as well in a normal person with directional dominance. This intangible tie rod should keep the nondominant eye "tracking" properly even if it were receiving no voluntary innervation. If, as I propose, only the total innervation to one eye need be recorded intracerebrally,[13] then only reflexive innervation need be sent to the other eye at all, and the voluntary innervation of the directionalizing eye may be the "something more" hinted at above as refining its aim. The perceptual mechanism may even be valuably assisted to ignore the innervations of the "nondominant" eye if these are merely in any way kept to a minimum, or particularly if they are restricted to the nonvoluntary ones, which do not have to tramp through the parietal or frontal cortex, shaking the ground of perception, on their way to the muscles.

Experimental tests of this idea should be possible to devise. For example, one could look to see whether the dominant eye starts to move a few sigmas before the nondominant eye when a voluntary movement is initiated from a stationary attitude of the eyes.

13. Not even all of the "tie rod" factors get into the record. Eye muscle innervations originating from vestibular stimulation, for example, do not. These are purposed to produce conjugate eye movements compensatory to head movements. This keeps the retinal image stationary on the retina so that the objects represented in it do not appear in motion. If, however, the eye movements occur without the head movements they are designed to cancel (as, for example, in caloric nystagmus), the movement of the retinal image over the retina causes apparent movement of the field: The perceptual homunculus, consulting the record of innervation, finds nothing there to tell him that the eyes are turning in exact compensation for the changing oculocentric direction of the objects in the moving retinal image. Supposing the eyes to be motionless, he can only conclude that the movement of the image connotes a movement of each object through space, and the individual therefore is caused to perceive a changing egocentric direction of the total field contents.

Against the idea, perhaps, is the fact that many persons (surely including some with dominance) have acquired an ability to move either eye alone at will. However, it is by no means certain that these actions can properly be considered positive and voluntary (Barrett,[14] Spiller,[15] Ayer,[16] Duke-Elder [17]).

IMPLICATIONS

Real understanding of the true nature of motor ocular dominance has been so long arriving that it will still be some time before the whole importance of the phenomenon can be evaluated. Even years ago, however (without anyone's knowing what motor ocular dominance really was), it was possible to determine that cross dominance of eye and hand may have great prognostic importance for speech and reading difficulties, personality aberrations, proneness to accidents and so on (Crider's review [18]).

For cross dominance to be a bad thing does not mean that ocular dominance per se is undesirable,[19] any more than it means that hand dominance is intrinsically bad. But it has been considered so by a few who have thought that the ideal stereoscopic visual system would necesarily have neither eye dominant. The very fact that ocular dominance is so common seems proof enough that it must be a good thing. In his careful study of 830 subjects submitted to a whole battery of sighting tests, Crider [3] found that 77.7 per cent had complete dominance of one eye, while only 7.35 per cent sighted about as often with one eye as with the other. If a person is better off for having a dominant eye, then it must, after all, be proper to say that the dominant eye is his better eye—not in the sense of affording a better-resolved mental image, but in regard to the subject's orientation in his visual environment.

It is generally considered that the incentive for the evolution of cerebral laterality—of which hand dominance is merely symptomatic—has been the enormous physiological complication created by the function of speech and the whole constellation of activities that involve symbolization (into which the hand becomes drawn in connection with writing). It has been mandatory for the control of all these activities to be centered in a single unpaired (hence unilateral) cortical territory in order for them to be properly controllable at all (Duke-Elder [20]).

Now, the peculiarly mammalian partial decussation of the optic nerves seems to have been an analogous attempt to put one hemisphere in control of the frightfully

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14. Barrett, J.: A Case of Voluntary Control of the Fusion Faculty, Med. J. Australia **1**:131, 1921.

15. Spiller, W. G.: Unilateral (Unassociated) Innervation of the Ocular Muscles, Tr. Am. Neurol. A. 1927, pp. 368-385.

16. Ayer, J. B.: Voluntary Dissociated Movements of the Eyes, J. Nerv. & Ment. Dis. **69**:69, 1929.

17. Duke-Elder, W. S.: Text-Book of Ophthalmology, vol. 4, St. Louis, C. V. Mosby Company, 1949, pp. 3998 and 4169.

18. Crider, B.: The Importance of the Dominant Eye, J. Psychol. **16**:145-151, 1943.

19. But, of course, it can be an unwitted source of inconvenience or exasperation if it is not catered to. Everyone should know which is his dominant eye, and be thereby governed in his arrangement of the layout of visual tasks. Crider [18] has suggested, for example, that a little study would show which side of her machine a typist should keep her shorthand notebook on when transcribing.

20. Duke-Elder,[17] pp. 3625-3628.

complex motor operation of both eyes in the interest of smooth binocular coordination. To give only the simplest possible example: Reflex coordinate movements of both eyes are caused by the arrival of excitation at one point in one area 17, whether the excitation that sets off a fixation reflex comes from both members of the pair of corresponding retinal points projecting there, or even from only one (either one) of them if the stimulus happens to be hidden from one eye by some obstacle.

Partial decussation, however, left the organism with a coequal pair of bilateral control systems, each centering in one hemisphere. This was as clumsy as the attempt to rule the complexity that was Rome with two consuls having identical jurisdictions. The development of the phenomenon of directional dominance seems to represent a further attempt to make partial decussation fulfil its destiny, by vesting the perceptionary employment of the motor properties of both eyes in a single center—"one sided," although not in respect to brain anatomy (as with writing-speech-hearing-reading symbol manipulation), but in respect to the left versus the right set of eye muscles and the bilaterally distributed nervous adnexa of one set. This view would be further strengthened if it should be found that voluntary control of eye movements applies to one eye only, according to my subsidiary hypothesis.

Tscherning, although he did not anticipate the present theory of directional dominance or suggest a steering gear mechanism, had a glimmering of both when he stated that dominance is evidence of a "tendency to a development of a uniocular vision in addition to the binocular vision." Speaking as a biologist, I would say that it seems as though the mammals are almost sorry that the original vertebrate constructed a pair of eyes instead of a single median one.[21] Certainly, for all the trouble the paired condition of the eyes has put them to, their only recompense is the stereoscopic three dimensionality of the binocular visual field. It is by no means certain that some vertebrates with totally decussated optic nerves do not enjoy that kind of vision anyway!

The theory of directional dominance, of course, has implications for the state of affairs in the "impartial-eyed" persons, who show no sighting preference. In them, the punctate locus of the binocular visual ego may be mobile and impermanent, or it may be in the median cyclopean position of Hering's "binoculus." But, at least, it is not permanently located at the perspective center of one eye. These persons do not, then, perceive the egocentric direction of every object or apparent object in haplopic or diplopic binocular vision, and in monocular vision with either eye, as if a dominant eye could see the object and as if it were the only eye that was seeing the object.

For the impartial-eyed subject the experiments diagrammed in figures 1 to 4, of course, will not hold. But, with the subject for whom they do work, the basis of his success can be described in two ways. First, he has a directional dominant eye; second, his space-perceptual apparatus "supposes" that what he is seeing with one eye is really being seen with the other—his brain is permanently deceived into

21. Earlier vertebrates did find it necessary to rearrange the paired brain roof eyes (represented in man only by the pineal gland). One member of each such pair was allowed to disappear, with the remaining member then secondarily moved into the sagittal plane of the head (Walls,[11] pp. 338-340).

thinking that the dominant eye is seeing even when it is not, so that the monocular sensations given by either eye are always referred to the dominant eye. But this is to say that the person who has a dominant eye cannot tell which eye is seeing when one eye is covered (except by the use of his "rational faculty"). A corollary is that those persons who do have "utrocular" vision, who can tell which eye is seeing when one eye is secretly covered, may be the impartial-eyed persons who have no directional dominance. It will be interesting to see whether an experimental study bears this out.

Chief of the practical implications of the directional theory of dominance are those relating to heterophoria and concomitant heterotropia. No matter how strong may be the "tendency" for the two eyes of a person to underconverge or overconverge for a given bifixation point, it is evident that he can always adopt such a posture, and one of his eyes can always adopt such an orbital posture as will allow that eye to fixate the point with exactly the same harmony of innervations playing on its muscles as would exist if he had no such tendency. Whatever the special pattern of tonuses in the muscles of the other eye may be that is required to overcome the abnormal tendency, no harm can be done to space perception if this special pattern is prevented from falsifying the perceptual direction of the point. If motor dominance is directional dominance, such prevention is automatic: Everything that is "special" about the eye muscle innervations in heterophoria would be concentrated on the muscles of the nondirectionalizing eye and never "recorded" at all. This is what has long been claimed,[21a] in the form of the dictum that "the whole of any heterophoria resides in the nondominant eye." The present theory would stamp approval on the practice of putting prismatic corrections exclusively in the lens before the nondominant eye whenever this is mechanically possible and cosmetically felicitous. This applies also to the related practice of placing Maddox rods and similar devices only in front of the nondominant eye in diagnostic procedures. To place any such optics over the one and only directionalizing eye would surely produce a temporary disorientation, expressing itself in manual and locomotor clumsiness, whereas to place it over an eye which is not concerned with giving direction could have no such effect.

The theory does not imply any necessary relation between the extent of heterophoria and the "strength" of dominance. We need reliable means, as objective as possible, for assessing the strength of dominance (which probably does occur in various degrees). But the heterophoria tests are unlikely to provide any. It would, however, be interesting to look for a relation between orthophoria and the absence of dominance—Crider's "impartial-eyed" condition. To suggest such a relationship may sound like suggesting the "extrapolation to zero" of a tie between heterophoria and strength of dominance, which I have just said probably does not exist. There is, however, a completely independent argument for a coupling of orthophoria and impartial eyedness. Under the direction theory, the phenomenon of dominance should serve as insurance against the tendency for a false projection to be caused by any abnormal innervation required to hold a heterophoric eye in team harness with its mate. It might be expected that in an orthophoric child

21a. Crider, B.: The Relationship of Eye Muscle Balance to the Sighting Eye, J. Exper. Psychol. **18**:152-154, 1935.

there would be less incentive to develop a dominant eye. If this is true, then the incidence of impartial eyedness should prove to be higher in the orthophoric adult population than in the heterophoric. This should not be difficult to check on.

In the case of any person with uniocular concomitant squint who once had had fusion, it would seem a certainty that the straight eye is one which had formerly been the patient's dominant eye.[22] Since a normal person prefers his dominant eye for monocular use, it seems inevitable that if a strabismic patient is to sacrifice one eye to amblyopia as his personal means of escape from diplopia, the eye he will keep in use will be the dominant eye if he has one. The case for the straight eye being the dominant (or ex-dominant) one in strabismus is even stronger than the case for the patient in the Maddox rod test having one eye that is "really straight" (even if the clinician cannot tell which it is). Moreover, the strength of the case does not actually depend on one's beliefs as regards the inner nature of the dominance phenomenon—it is enough that dominance expresses itself in a monocular sighting preference.

If, now, the relationship of uniocular squint and dominance is "extrapolated," we find ourselves faced with the postulate that a person with alternating squint has no dominant eye and has alternating squint because he never had one. The direction theory does seem to imply that if an impartial-eyed person becomes a squinter he will have no reason permanently to surrender the ability to fixate with one of his eyes, since he could momentarily suppress either and still directionalize with the other. But if there were a great difference in visual acuity between his two eyes, he might find more advantage in being uniocular and always enjoying good acuity than in being able to fixate with either eye but having low acuity half the time.

I do not know that there are adequate data, already available, from which to work out a settlement of this matter. The percentage of patients with concomitant squint who are alternators should not be less than the percentage of impartial-eyed persons in the general population, if the person with alternating squint is to be interpreted as one who had never acquired motor ocular dominance. Duke-Elder [23] gives only some rather old figures, which make the incidence of alteration in concomitant squint 11 per cent. Crider,[3] in one table, gives 7.35 per cent as the incidence of impartial eyedness, but elsewhere in the same paper he states that 27.75 per cent of males and 33.15 per cent of females are impartial eyed. On the other hand, Duke-Elder's authority (Lagleyze) found that 10 of 11 persons with alternating squint had equal visual acuities in the two eyes, while three of four with uniocular squint had unequal visual acuities. "Unequal" on how finely graduated a test chart? "Equal" within what limits of tolerance? One may even wonder how anyone with uniocular squint can avoid having unequal visual acuities. But the basic question raised by these meager data is whether patients with alternating squint alternate only because they have equal acuities, and not because they have

22. This is not to be confused with the common statement that the straight eye is the dominant eye. Such a statement assumes that the better eye is the dominant eye even in a nonsquinter and overlooks the fact that dominance is a phenomenon of binocular vision. No uniocular person, strabismic or *borgne*, can be said to have a dominant eye at all.

23. Duke-Elder,[17] pp. 4006-4007.

never had a dominant eye. Is the percentage of persons with alternating squint having equal visual acuities really higher than the incidence of equal acuities in the general population—with all testees given the same acuity test?

It remains conceivable that a developing squint is influenced to become alternating or uniocular by the matter of whether or not the patient has theretofore had a perceptually dominant eye (by the criteria of rivalry and phi dominance, etc.). Nor should it be lost sight of that there are many who believe that everyone with concomitant squint starts out with alternating squint—a concept which, if true, would stop all talk about dominance in this connection.

Another implied relation between visual acuity and directional dominance inheres in the recommendation so many authorities make to the refractionist, to undercorrect the nondominant eye so as not to permit its visual acuity corrected to become as high as that of the dominant eye. I do not see that my theory implies either wisdom or unwisdom in this recommendation, or that the theory is weakened if it is true that to allow the nondominant eye to have the higher acuity is a bad thing. The perceptual resolution of an object will be that given by the better eye, and it should not matter whether this eye or the other is the one that gives direction. I strongly suspect that higher acuity corrected in the nondominant eye is not a bad thing. The matter should not differ from that for persons who wear no spectacles at all; and, as noted above, Gahagan found many emmetropes with higher acuity in the nondominant eye but mentions no sort of complaint by this group.

It is possible that in testing for monocular visual acuity the nondominant eye always has slightly depressed acuity because fixation of the target is less steady with it. Haploscopic charts which offer dummy targets to the eye not being tested, such as are used in the Ortho-Rater, would perhaps eliminate this; but the effect would be masked by the difference in acuity shown by any eye, depending on whether the other eye is being given something similar to look at, or not. It is not even known whether the "physiological nystagmus" has any greater amplitude and/or frequency in a nondominant eye than in its dominant mate. Here, then, is still another investigation suggested by our prolonged stare, here, at the phenomenon of ocular dominance.

There are recommendations that might be made concerning the design of binocular optical instruments in the light of the direction theory. One such instrument is the stereoscopic rangefinder (or heightfinder). In these instruments as they are built by most or all makers, the range taker's adjustment of the "measuring knob" eliminates the retinal disparity of the images of the target and the reticle by moving the image in the right eye to the left or to the right. In the use of the instrument, the left eye is allowed to remain in the primary position, while the right eye is called on to make all of the changes of vergence. It should make a difference, in the range taker's performance, whether he is of the right-eyed majority or is, instead, left eyed. No similar considerations are involved in the design of such instruments as monobjective binocular microscopes, in which the eyepieces are geared together so that both eyes are equally charged with any change of vergence required to adjust for the interpupillary distance. But in the use of haploscopic instruments for some kinds of visual research, any such coupling of the eyepieces would be highly undesirable. When haploscopes in the form of amblyoscopes are

used for orthoptic training, I believe it needs to be considered whether the dominant eye might not best be allowed to be motionless, with all "muscle exercises" (really, brain exercises!) given only to the other eye.

SUMMARY AND CONCLUSIONS

There are at least two kinds of ocular dominance, one of which has a motor character and expresses itself in monocular sighting preference but is independent of the brainedness laterality which governs handedness.

It is hypothesized that motor ocular dominance consists in the employment of the patterns of innervation sent to the rotatory muscles of one eye only, for the establishment of the perceived egocentric directions of visual objects in either binocular or monocular vision.

The various reliable criteria of motor ocular dominance are shown to be asymmetrical phenomena which, under the hypothesis of directional dominance set forth in this study, would be expected to occur.

It is further suggested that in voluntary eye movements, voluntary innervation may normally go to the muscles of the directionally dominant eye only, the other eye being caused to "track" by a constellation of well known reflexes and synkineses.

Some nonclinical and clinical implications of the theory of directional dominance are outlined and discussed, and several subsidiary problems, raised by the theory, are pointed out, the solutions of which would afford further tests of the validity of the theory.

IRIS CYST FOLLOWING TRAUMATIC IMPLANTATION OF GLASS SPLINTER

With Foreign Body Probably Retained in Ciliary Area Forty-Five Years

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THE LITERATURE contains many reports of traumatic cyst of the iris caused by the implantation of glass or other substances in the eye. Among others who have published articles on this subject is Perera,[1] who in 1937 read a paper before the American Academy of Ophthalmology and Oto-Laryngology on epithelium in the anterior chamber of the eye after operation and injury.

On Nov. 28, 1928, I [2] presented before this Section a case in which a fragment of glass had been embedded in the eye for 28 years. In this case a high school student was experimenting in the laboratory with a mixture of sulfuric acid and potassium nitrate in a glass retort, placed over a Bunsen burner; its explosion caused a fragment of glass to penetrate the periphery of the eyeball and become embedded in the retina. The incised scleral wound was closed with three episcleral sutures. This procedure was followed by a hemorrhage in the vitreous. When the blood had been absorbed, a glistening, reflecting, yellowish line could be seen in the retina. This line, which was visible in the periphery of the fundus under the incised wound, was identified as a fragment of glass. The vision and fundus remained normal during the 28 years.

This case, together with many other recorded instances, proves that internal tissues of the eye can tolerate the presence of glass for many years provided no infection or local irritation occurs. The case which follows presents several interesting clinical features.

REPORT OF A CASE

W. A., a man aged 51, a concert pianist, was in excellent health when seen by me.

In 1902, when he was 3½ years old, he was playing with other children in the dining room of his parents' home in Utrecht, Netherlands, when he was struck in the right eye with a fragment of a glass plate, which penetrated the eyeball. The child was taken by his mother to the family ophthalmologist, who applied local treatment to the eye; he stated that he saw a piece of glass over the site of the injury, and he cautioned the mother not to allow the glass to be removed unless complications arose requiring further treatment. Accordingly, no attention was paid to the ocular injury, nor was any inconvenience noted for many years. I have been unable to obtain any details of the accident from attending ophthalmologists, as all records were destroyed during a bombardment of Utrecht in 1940.

Presented before the Section of Ophthalmology, New York Academy of Medicine, Nov. 20, 1950.

1. Perera, C. A.: Epithelium in the Anterior Chamber of the Eye After Operations and Injury, Am. J. Ophth. **21**:605-617 (June) 1938.

2. Cohen, Martin: Glass Embedded in the Eye, Arch. Ophth. **1**:528-529 (April) 1929.

In 1937 the patient consulted the mother's ophthalmologist in Utrecht because the eye injury had been responsible for his rejection for service in the Netherlands army. Examination of the eye at that time disclosed a scar near the corneal limbus caused by the penetration of glass into the globe. The patient was again advised not to have the glass removed, as the eye was apparently free from inflammation and its removal in this location might endanger the normal, left eye. A similar opinion was expressed by another ophthalmologist in consultation. The patient then left the Netherlands and came to the United States, where he remained for many years. During this period he had three mild attacks of conjunctivitis, all of short duration and separated by long intervals. During most of this period the right eye remained free from inflammation.

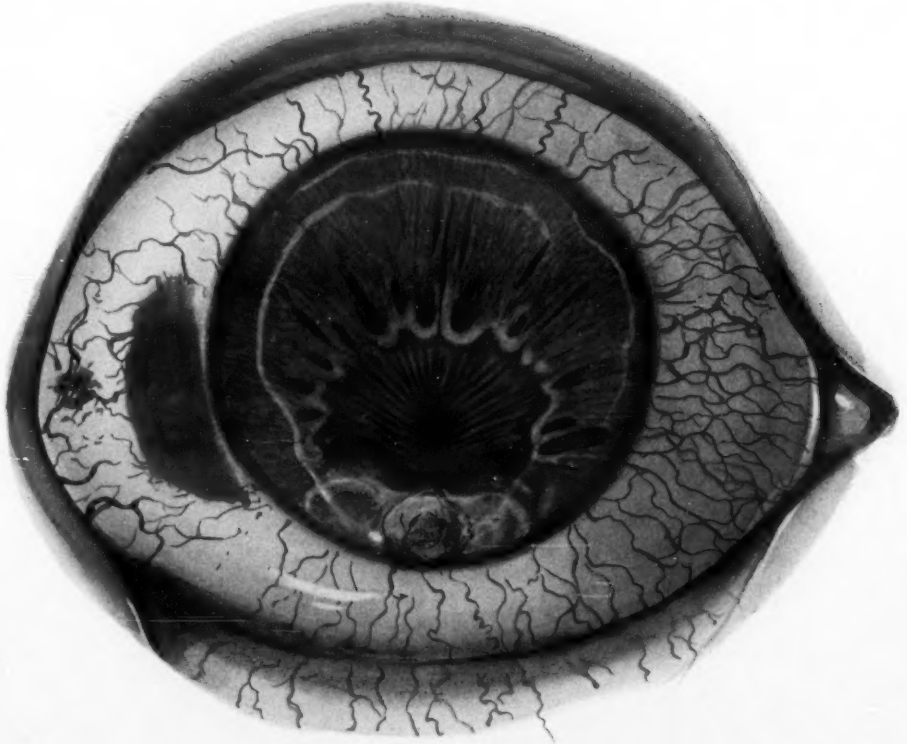
On Feb. 2, 1950 he suffered an acute attack of severe headache and radiating pain from the eye; the following day he consulted me for the first time in regard to his ocular condition. At this time the left eye was normal; the right eye was extremely painful. Intraocular tension was greatly increased; light projection was normal; vision was 20/200 without correction. A fresh conjunctival hemorrhage was noted near the nasal limbus. Extensive circumcorneal ciliary injection was evident, and a linear scar, 5 mm. in length, was situated near the corneal limbus. The anterior chamber was shallow, and the pupil was slit shaped, owing to a posterior synechia which had formed as the result of an exudate visible at the lower extremity of the pupil. A multilocular cyst was seen in the lower sector of the anterior chamber close to the cornea. Slit lamp examination of the iris and cornea revealed that the central cyst was translucent and its posterior wall vascularized. The adjacent nasal and temporal cysts were also translucent and were capped with a pigmented crescent. A small circular perforation was seen in the cyst located nasally. Two degenerated and undulating folds of Descemet's membrane were situated in close proximity in the area of the arcus senilis. The degenerated cysts of the iris originated in the region of the scar; there an ingrowth of probably epithelial cells had migrated and proliferated in the anterior chamber to form iris cysts; when these cysts first became visible could not be determined. Biomicroscopic examination showed no membrane on the posterior surface of the cornea or the surface of the iris. Gonioscopic study was not available. The fundus appeared normal.

The diagnosis was acute congestive glaucoma (probably secondary).

The acute glaucoma was probably secondary, for the following reasons: It is known that implantation cysts progress very slowly over many years, causing no acute symptoms until the terminal stage of the cyst, when it produces obstruction in the peripheral angle of the anterior chamber, leading to symptoms of secondary glaucoma. In primary (acute congestive) glaucoma the cyst is observed mainly in the late stages of the disease and usually involves both eyes and is accompanied with optic nerve atrophy. The latter signs were not observed in this case. The status of the patient's right eye at the time of examination was completely satisfactory with respect to the course of the acute glaucoma. The rapid course of the attack was the basis for my inclination to look on the glaucoma as secondary to a long-standing pathologic condition in the eye.

The acute glaucoma was treated by the instillation of 2 drops of 2 per cent pilocarpine nitrate into the eye every two hours. Hot compresses, 10 grains (0.65 Gm.) of acetylsalicylic acid three times a day, laxatives and rest were prescribed. After two days of this treatment the ocular pain was greatly relieved and the ocular tension, though not normal, was reduced to 35 mm. of mercury. The ciliary injection was less evident, and the vision had improved to 20/30 without any correction by the sixth day. The patient was also advised to continue the use of pilocarpine drops to prevent, if possible, a recurrence of the glaucoma. He was slightly myopic. There existed a partial blocking of the peripheral angle of the anterior chamber, produced by the multilocular cyst; this condition at times caused serious ocular complications. I therefore advised the patient to have a periodic ophthalmic examination to insure precautionary measures in the treatment of this eye. After the first ophthalmic examination kodachrome® films of the outer segment of the eye were taken. An accurate colored drawing from these films is presented.

It is well known that glass particles in the eyeball can frequently be demonstrated with the Vogt method of roentgenography, by applying a dental plate at the corner of the orbital cavity. The patient, however, did not avail himself of



Outer segment of the eye, showing implantation cyst of the iris (drawing from kodachrome® film).

this diagnostic aid, and it is therefore difficult to state with certainty that a fragment of glass is still present in the eye, though it is highly probable.

Several surgical procedures have been advocated for the treatment of implantation cyst, such as (1) removal of the cyst, or (2) aspiration of the contents, followed by injection of a few drops of iodine into the cyst. Roentgen therapy has given satisfactory results when the radiation is administered in doses small enough to avoid a complicated cataract; this treatment was recommended to the patient.

SUMMARY

A case is reported of an injury resulting from a fragment of glass which had probably been retained in the eye for 45 years without causing deterioration of vision or changes in the fundus.

In this case, the location of the scar, near the lower corneal limbus, represented the site where the glass had probably perforated the globe 45 years earlier.

Implantation of the iris cyst is due primarily to the proliferation and migration of epithelial cells from the site of the perforation into the anterior chamber and iris.

The secondary acute congestive glaucoma in the present case was of short duration and had had no effect on vision or the fundus after a lapse of six weeks, when the patient was last examined.

Treatment was solely directed toward relief of the acute glaucoma.

The noninflammatory reaction of glass in the eye is due to the fact that approximately 98 per cent of glass is made up of neutral sodium silicate, which is chemically inert.

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POSTDIPHTHERITIC CYCLOPLEGIA

Report of Two Cases

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ANDERSON, S. C.

A REVIEW of the available literature reveals that in recent years few cases of postdiphtheritic cycloplegia have been reported in this country. This is no doubt due in large measure to the increasing use of diphtheria prophylaxis. During the course of World War II there was a flurry of reported cases of diphtheria in the armed forces, with varying percentages of cycloplegia.

Delp, Sutherland and Hashinger [1] reported five cases of postdiphtheritic polyneuritis with albuminocytological dissociation, in four of which there was paralysis of accommodation. Copsey [2] reported 17 cases of postdiphtheritic paralysis, only four of which were described in detail, and in three of these there was a complaint of blurred vision. Hertz and Thygeson,[3] reporting on 403 cases of diphtheria, found neurological complications in 144. Of their 144 cases of postdiphtheritic paralysis, paralysis of accommodation developed in 64, or 44.4 per cent, (15.8 per cent of the total number of cases). They stated that accommodative paralysis is next in frequency to palatal paralysis. The latency was from three to five weeks, and the duration was generally less than five weeks. It was their opinion that the paralysis will run its inevitable course in spite of all treatment and that neither vitamin B nor the injection of diphtheria antitoxin is of any value after the paralysis has begun. Gaskill and Korb,[4] reporting 140 cases of cutaneous diphtheria, found accommodative paralysis in 20, or 14.3 per cent. Givhan and Mahon [5] reported 10 cases of diphtheria, in three of which blurring of vision was a complication.

In May 1949 each ophthalmologist in the State of South Carolina was queried as to how many, if any, cases of cycloplegia he had seen during his years of practice. Replies were received and tabulated showing that a total number of five cases had been encountered. Personal communications from ophthalmologic friends connected with the Massachusetts Eye and Ear Infirmary, the Wilmer Ophthalmological Institute and the Grady Clay Memorial Clinic, Atlanta, Ga., indicated the paucity of cases in the records of these institutions.

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1. Delp, M. H.; Sutherland, G. F., and Hashinger, E. H.: Ann. Int. Med. **24**:618-628 (April) 1946.

2. Copsey, H. G.: M. Clin. North America **30**:445-450 (March) 1946.

3. Hertz, M., and Thygeson, P.: Acta psychiat. et neurol., 1947, supp. 44.

4. Gaskill, H. S., and Korb, M.: Occurrence of Multiple Neuritis in Cases of Cutaneous Diphtheria, Arch. Neurol. & Psychiat. **55**:559-572 (June) 1946.

5. Givhan, E. G., Jr., and Mahon, E. J.: U. S. Nav. M. Bull. **45**:267-269 (Aug.) 1946.

REPORT OF CASES

CASE 1.—P. C., a white schoolgirl aged 11, was referred by Dr. J. P. Booker, of Walhalla, S. C., on Dec. 15, 1948. The mother stated that the child became ill with sore throat on November 11 and was in bed 10 days. Her condition was diagnosed as diphtheria by her family physician, and she was given diphtheria antitoxin and penicillin. She returned to school on December 6. The patient stated that she could read for the first week of her illness but that she had not been able to read since the second week. She lost her voice after four days and was unable to speak above a whisper for 12 days.

Physical examination revealed a healthy-appearing, well nourished girl. The temperature was normal; the pulse rate was 105; the heart sounds were normal; the weight was 67 pounds (30.4 Kg.), and the height was 56 inches (142 cm.). She felt well, was going to school daily and had no complaint other than the inability to read. External examination of the eyes revealed nothing abnormal; the conjunctivas were clear and white, and the cornea, anterior chamber, iris and pupil were normal. The pupils reacted amply and equally to light directly, as well as consensually. The accommodative reaction was somewhat retarded, but this was not noted until an object was brought to within about 135 mm. The near point of convergence was 70 mm. The media and fundi were not remarkable. Vision in each eye separately was 20/100, and with the Maddox rod 16 Δ of esophoria was measured. With the Prince rule she could not make out any of the letters at 50 cm. with either eye. At 33 cm. the larger letters on the rule could be made out with a + 5.00 D. sphere before each eye. At the trial case, with a + 2.50 D. sphere before each eye, she could read 20/20 Snellen type, and with a + 2.50 D. addition she could read Jaeger type 3 with each eye. Instillation of 2 per cent homatropine hydrobromide solution was made three times at five minute intervals, after which the trial case corrections were the same. Findings on retinoscopic examination were practically the same. A prescription of + 2.50 D. sph., add + 2.50 D. sph. was given for each eye. She was able to read and to return to school.

The patient was seen again on April 2, 1949, when she stated that she had been unable to read without her glasses until February 15, since which time glasses had been unnecessary. On this date the vision was 20/20 in each eye uncorrected. With homatropine refraction, she accepted + 2.00 D. sph. $\bigcirc$ + 0.50 cyl., axis 90 for the right eye and 2.50 D. sph. $\bigcirc$ + 0.50 cyl., axis 90 for the left eye. It was concluded that she had had a cycloplegia of 2½ months' duration after diphtheria.

CASE 2.—L. H., a white schoolgirl aged 9, was first seen on Nov. 23, 1948, having been referred by Dr. Hubert Milford, of Hartwell, Ga. There was a history of sore throat for the past 24 hours, together with malaise and fever.

Examination revealed a well nourished girl, with a temperature of 99.4 F. The tonsils were acutely inflamed and covered with a grayish yellow membrane, almost meeting at the midline. The cervical lymph nodes were enlarged and nodular. The patient was admitted to the Anderson Memorial Hospital, and smear and culture of the tonsillar membrane yielded Corynebacterium diphtheriae. She was given 20,000 U. S. P. units of diphtheria antitoxin. The diphtheria rapidly improved and she was allowed to leave the hospital on November 28. On December 29 she returned with the history of having been unable to read since she left the hospital. External examination of the eyes revealed nothing abnormal, the pupils being round and equal and reacting normally to light. The records contained no reference to reaction to accommodation. The media and fundi were normal; vision in each eye uncorrected was 20/100, the Jaeger reading not being obtained. With the trial case, a correction of + 1.50 D. sph. $\bigcirc$ + 0.50 cyl., axis 90 for each eye gave vision of 20/20, and with the addition of a + 2.00 D. sphere for each eye she could read Jaeger type 3. A 2 per cent homatropine hydrobromide solution was instilled into each eye at five minute intervals three times; after instillation of the drops she accepted the same correction at the trial case as before. Finding on retinoscopic examination were essentially the same. She was given bifocal glasses with the following correction for each eye: + 1.50 D. sph. $\bigcirc$ + 0.50 cyl., axis 90, add + 2.00 D. sph. At this time it was noted that her voice had a nasal tone, indicating partial palatal paralysis. On January 15 she returned, stating that she had been wearing her glasses constantly and that she could read very well with them, and not at all without them.

The pupils were round and equal and reacted normally to light and in accommodation. On January 18 she was again seen, at which time uncorrected vision in each eye was 20/20, with ability to read Jaeger type 1; she was instructed to leave off her glasses. A diagnosis of cycloplegia following diphtheria was made, and the duration of the cycloplegia in this case was estimated at seven weeks.

SUMMARY

Two cases of postdiphtheritic cycloplegia are reported, in one of which the paralysis lasted 2½ months and in the other seven weeks. Bifocal lenses were prescribed in each case and were worn with satisfaction. Recovery was complete and uneventful in each case.

Professional Bldg.

ROSE BENGAL TEST IN DIAGNOSIS OF DEFICIENT TEAR FORMATION

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PHILADELPHIA

IN RECENT years more attention is being given to a group of conditions associated with deficient tear formation, or hypofunction of the lacrimal gland. The cause or causes of these conditions are as yet unknown. Whether deficiency in tear formation is caused by endocrine disturbances, infection, the aging process or other factors remains to be determined. It is probable that no one etiological agent is responsible.

Sjøgren introduced the term "keratoconjunctivitis sicca," but in English-speaking countries "keratitis sicca" has become the more prevalent name for the disturbance. Emphasis has been laid mostly on the corneal changes associated with the severer forms of the disease. Consequently, many cases of the milder form involving only the conjunctiva have been overlooked. The purpose of this paper is to describe the rose bengal test for the conjunctival, as well as the corneal, changes associated with deficient tear formation. This test is relatively unknown in this country but appears to be worth while.

Schirmer,[1] the originator of the well known Schirmer filter paper test for tear formation, also introduced the aniline dye rose bengal as a suitable agent for showing the conjunctival changes accompanying deficient tear formation. Kleefeld[2] recommended a 1 per cent rose bengal solution for vital staining of the cornea and conjunctiva.

Clinically, vital staining of the cornea and conjunctiva is of only theoretical interest. The process involves staining of living cells and is of importance in experimental work only. It is accomplished by repeated instillations of an aqueous solution of an aniline dye into the conjunctival cul-de-sac and is a painful procedure.[3] Surface staining, however, is of great practical importance. The ordinary fluorescein test for denudation of the corneal epithelium is an example of surface staining. Other dyes can be used. Sjøgren[4] recommended surface staining with a 1 per cent aqueous solution of rose bengal in the diagnosis of keratoconjunctivitis sicca. After a drop of this solution has been instilled into the conjunctival cul-de-sac being allowed to remain for 15 seconds or more and the excess dye removed by irrigation, devitalized epithelial cells and mucus retain the rose stain. All mucus should be removed from the cul-de-sac before instillation of the rose bengal for

1. Schirmer, cited by Sjøgren, H.: Zur Kenntnis der Keratoconjunctivitis Sicca (Keratitis filiformis bei Hypofunktion der Tränendrüsen), Acta ophth., 1933, supp. 2, pp. 1-151.

2. Kleefeld, G.: Une nouvelle coloration des ulcères coréens, Bull. Soc. belge d'opht. **41**:58, 1920.

3. Berliner, M. L.: Biomicroscopy of the Eye: Slit Lamp Microscopy of the Living Eye, New York, Paul B. Hoeber, Inc., 1943, vol. 1.

4. Sjøgren, H.: Keratoconjunctivitis Sicca, in Ridley, F., and Sorsby, A.: Modern Trends in Ophthalmology, New York, Paul B. Hoeber, Inc., 1940, p. 404.

clinical testing. With a high power corneal microscope (magnification, 40 ×) the stained areas are seen to consist of faintly outlined epithelial cells with deeply colored nuclei. Bowman's membrane, the corneal stroma and subepithelial conjunctival tissue are not stained by rose bengal, whereas they do take a fluorescein stain.

THE ROSE BENGAL TEST

Rose bengal is obtainable from several chemical manufacturers [5] and can be made up in solution by any pharmacist very easily. It may be used in the conjunctival cul-de-sac in a concentration of 1 per cent in isotonic sodium chloride solution or in 1:5,000 benzalkonium chloride U. S. P. (zephiran® chloride) solution for more effective maintenance of sterility. Tests indicate that benzalkonium chloride in this concentration does not interfere with the acceptance of the dye by devitalized epithelium or produce false positive tests by injuring the epithelium. Instillation of this dye is a somewhat uncomfortable procedure, but the irritation wears off after removal of the excess dye by irrigation. The discomfort seems to be roughly proportional to the amount of stained epithelium and, if severe, can be relieved quickly by topical application of tetracaine (pontocaine®) hydrochloride. Tests show no false positive rose bengal tests from use of tetracaine for local anesthesia prior to the instillation of the dye, and employment of this anesthetic may be routine, thereby avoiding subjective irritation. Epithelium which takes the stain remains rose colored for several hours, so that with strongly positive staining responses dark glasses may be of cosmetic value after instillation of the dye.

In eyes with marked deficiency of tear formation, rose bengal stains the bulbar conjunctiva and caruncle intensely in the exposed area between the lids. In cases of corneal involvement the epithelial filaments also take the rose stain, whereas the areas of Bowman's membrane from which the filaments of epithelium have come loose do not. The bulbar conjunctiva below the limbus down into the cul-de-sac is usually stained, as is occasionally the palpebral conjunctiva of the lower lid. If a Schirmer test is done just before the rose bengal test, the conjunctival epithelium in the area rubbed by the paper will be damaged and subsequently take the rose bengal stain. Hence staining of the conjunctiva with rose bengal only in the area of previous contact with the filter paper should not be construed as diagnostic of deficient tear formation. Staining of the palpebral conjunctiva of the upper lid or the part of the bulbar conjunctiva which is normally covered by the upper lid has not been seen. A likely reason for this is that these areas are the moistest and the least exposed to evaporation of what tear fluid is present. Exposure appears to be a definite factor in determining which areas of the conjunctiva show changes earliest in mild cases of tear deficiency. In recording the intensity of rose bengal staining of devitalized epithelium, a simple scheme, such as that shown in the tabulation, may be employed.

Strongly positive	Intense staining of bulbar conjunctiva with or without corneal staining
Moderately positive	Staining of most of exposed bulbar conjunctiva between lids and inferior cul-de-sac
Negative	No staining, or scattered spotty staining visible only with slit lamp

5. Rose bengal powder was obtained from Arthur H. Thomas Company, West Washington Square, Philadelphia.

EVALUATION OF ROSE BENGAL TEST

I have found only one article in the literature which has reported the diagnostic use of rose bengal to any great extent in cases of keratoconjunctivitis sicca. Stenstam [6] investigated the occurrence of keratoconjunctivitis sicca among 494 hospitalized patients with rheumatoid arthritis and rheumatic fever in Sweden. This paper is also the only one in the literature on keratoconjunctivitis sicca from the point of view of the rheumatologist, although it is known that the arthritic patient frequently has a "dry eye." It seems that this manifestation of rheumatoid disease has been generally overlooked by those interested in arthritis. Stenstam found that 10 per cent of his arthritic patients had keratoconjunctivitis sicca, i. e., conjunctival and corneal manifestations with strongly positive rose bengal and Schirmer tests. He also found that another 15 per cent of these patients had some deficiency in tear formation, as indicated by conjunctival staining with rose bengal and positive or negative Schirmer tests. Since the age range of his material was limited to from 12 to 55 years, senility was thought not to be an important cause of reduction in lacrimal gland function in the patients studied. Symptoms of deficient tear formation did not appear usually until the age of 40 and were seen mainly in arthritic patients who had had the disease for years. The joint symptoms commonly preceded the ocular symptoms. His incidence of keratoconjunctivitis sicca of 35 per cent in men and 65 per cent in women arthritics is at variance with the sex incidence reported elsewhere. The over-all sex incidence according to other investigations is 86 per cent for women and 14 per cent for men (Henderson [7]).

The standard test for deficient tear formation in present clinical use is the Schirmer test. Whatman no. 41 filter paper is cut into strips 5 mm. wide and 35 mm. long. A crease is made in the paper 5 mm. from one end, and the bent end of the paper is placed between the lower lid and the bulbar conjunctiva at the junction of the middle and the inner third of the lower lid. Schirmer's standard of 15 mm. of wetting in five minutes for normal eyes has been accepted for years. The test must, of course, be performed before any manipulation of the eyes which might produce tearing. Every one who has used this test to any extent has realized that it is inaccurate in estimating the amount of tear flow except in an advanced case of keratoconjunctivitis sicca. The amount of wetting of the filter paper is influenced by the amount of blinking of the eyes during the five minute test. The irritating effect of the paper on the conjunctiva may stimulate more tear formation than is normally present when the lacrimal gland has partial function. There is no real agreement as to what constitutes a normal reading, though it is generally considered that in older people, particularly women, some decrease in tear formation as measured by the Schirmer test is physiological.

In an effort to find a more reliable test for indicating particularly the early cases of deficiency in tear formation, I have had the opportunity to compare the rose bengal test which Stenstam [6] has used with the Schirmer test in a hospitalized group of arthritic patients, who might be expected to have keratoconjunctivitis sicca in all degrees of severity. This study was made primarily at the Philadelphia

6. Stenstam, T.: On Occurrence of Keratoconjunctivitis Sicca in Cases of Rheumatoid Arthritis, Acta med. Scandinav. **127**:139-148, 1947.

7. Henderson, J. W.: Keratoconjunctivitis Sicca, Am. J. Ophth. **33**:197-223, 1950.

Home for Incurables, where there are a number of patients with advanced rheumatoid arthritis, many of whom have ocular complaints and present an ideal opportunity for this study. Additional cases were included from private practice and from hospital eye clinics in Philadelphia.

In order to compare the rose bengal test with the Schirmer test, the results obtained with 98 eyes were analyzed. Both tests were done at the same examination, the Schirmer test being made first. All patients for whom tests are tabulated showed clinical improvement with use of methyl cellulose drops or artificial tears. Symptomatic relief, though an inaccurate means of classifying patients, was the only possible criterion in deciding in mild cases whether or not to make the diagnosis. Three patients had definitely positive rose bengal and Schirmer tests but no symptoms of deficient tear formation. The criteria used for determining

TABLE 1.—*Criteria of Severity of Deficiency of Tear Formation as Determined by the Rose Bengal and Schirmer Tests*

	Rose Bengal Test	Schirmer Test
Strongly positive	Intense conjunctival staining with or without corneal staining	0- 3 mm.
Moderately positive	Less intense staining of bulbar conjunctiva	4-10 mm.
Mildly positive	Patchy staining of bulbar conjunctiva	11-15 mm.
Negative	No grossly visible staining	More than 15 mm.

TABLE 2.—*Results of Rose Bengal and Schirmer Tests of Tear Deficiency*

	Rose Bengal Test	Schirmer Test
Strongly positive	16	31
Moderately positive	49	30
Mildly positive	30	14
Negative	3	23
Total no. of eyes	98	98

the severity of tear deficiency are of necessity arbitrary, but those listed in table 1 were employed.

Allowances were made for epithelial damage from the filter paper, which frequently produces a well circumscribed area of staining on the inferior bulbar conjunctiva when the rose bengal test is done immediately after the Schirmer test. The results of the two tests are to be found in table 2.

There was good correlation between the Schirmer and the rose bengal test in the cases of definitely deficient tear formation, as judged by the eyes for which the test was moderately or strongly positive. The important conclusion to be derived from these figures concerns the milder cases of the disease. Thirty of the eyes showed mildly positive rose bengal tests, and for 23 of them the Schirmer test was negative. Three eyes had negative rose bengal tests. These were the unaffected eyes in patients in whom only one eye seemed to be involved at the time. Though the number of cases is not very large, it seems that the rose bengal test is more likely to be definitely positive than the Schirmer test in the early or mild cases. It is a patient with a mild deficiency in whom the diagnosis is most likely to be missed. A

total of 35 patients with keratoconjunctivitis sicca are included in this study, 19 of whom may be considered to have mild deficiency in tear formation.

There seems to be no doubt that deficient tear formation will produce devitalized conjunctival epithelium which takes the rose bengal stain. There was no case in this series in which the Schirmer test was positive, i. e., wetting of 15 mm. or less, and the rose bengal test negative. On the other hand, 20 eyes for which the rose bengal test was positive had negative Schirmer tests at the same time. Each of these patients complained of symptoms which the patient believed were relieved by drops of methyl cellulose. In 33 control patients, of both sexes and with a range in age of from 13 to 87 years, the rose bengal test was negative. These patients had various ocular diseases other than keratoconjunctivitis sicca or had no ocular disease.

Four patients were seen who had positive rose bengal tests but who did not have keratoconjunctivitis sicca. One was a man, aged 80, with epithelial dystrophy of the cornea. The test was positive only for the cornea. Another was a woman, aged 70, with redundant bulbar conjunctiva below the limbus in each eye. Her somewhat spastic lower lids rubbed the loose conjunctiva into horizontal folds, the ridges of which stained with rose bengal, presumably because of epithelial damage. In a similar case the condition was due to trichiasis, and a fourth patient, with an acute conjunctival and corneal lime burn, showed intense staining with rose bengal in the burned area.

COMMENT

The diagnosis in a mild case of keratoconjunctivitis sicca has of necessity been made primarily on the basis of the patient's subjective complaints, mild or moderate decrease in tear formation as measured by the Schirmer test and the therapeutic response to tear substitutes. These criteria are, of course, inaccurate in many instances. It seems that the rose bengal test, which is objective, and apparently more likely to be positive in mild cases than is the Schirmer test, is a worth while aid in arriving at this diagnosis. The intensity of the staining with rose bengal is variable even in advanced cases, but a grossly visible staining reaction is easier to interpret than is the amount of wetting of a piece of filter paper, subject to the several inaccuracies previously referred to. The rose bengal test as described is not entirely specific for deficient tear formation. The dye appears to stain selectively devitalized conjunctival and corneal epithelium, for some unknown reason. Disease or injury causing such epithelial damage will result in a positive rose bengal test. However, the causes of a positive test other than deficient tear formation are usually obvious. Several have been referred to earlier in this paper. A number of patients, who had conditions with symptoms similar to those of deficient tear formation, and were included among the controls, had negative rose bengal tests. In the absence of an obvious cause for epithelial damage, a positive rose bengal test should indicate consideration of the diagnosis of keratoconjunctivitis sicca as an underlying, if not the sole, factor in the condition in question. As with most diagnostic tests, there are advantages and disadvantages. The important advantage of the rose bengal test over the Schirmer test is the apparent greater reliability in cases of mild deficiency in tear formation. One disadvantage is that unless instillation is preceded by tetracaine anesthesia it is somewhat uncomfortable, particularly in eyes that show a positive response. This discomfort, however, can be avoided by preliminary topical

anesthesia, which is recommended as a routine procedure, without danger of producing false positive tests. If the test is positive, the bulbar conjunctiva retains the stain grossly for several hours after instillation of the dye, so that dark glasses may be needed for cosmetic reasons. However, the clinical establishment of the diagnosis, the therapeutic relief afforded by methyl cellulose drops or an equivalent and the gratitude of the patient, who frequently has consulted several ophthalmologists before the diagnosis is made, should certainly outweigh the minor disadvantages mentioned. It is worth while to emphasize here that the staining reaction with rose bengal is not apparently affected by use of methyl cellulose or any other artificial tear preparation. These substances afforded symptomatic relief but, of course, have no curative effect on the underlying tear deficiency.

SUMMARY

The rose bengal diagnostic test for deficient tear formation (keratoconjunctivitis sicca) is described. It is a test only rarely used in this country, judging by reports in the literature, but is better known in Scandinavia.

A comparative study of the rose bengal test and the Schirmer test has been made on 98 eyes with deficient tear formation.

The rose bengal test seems to be more frequently positive in mild cases of keratoconjunctivitis sicca than is the Schirmer test and should be an aid in the diagnosis of early forms of this disease.

A study of the specificity of the rose bengal test has been made.

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PERIPHERAL FUSION

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THE CONCEPT of peripheral fusion was first introduced in ophthalmology by Hermann Burian.[1] He demonstrated that identical objects imaged on peripheral disparate points of the retina can induce a fusional movement of the eyes which brings the images on corresponding points. The peripheral fusion may even be so strong as to break the fusion of images situated on the macular region; e. g., a point fixated with both eyes may be seen as two points when the periphery of the eyes is stimulated haploscopically by disparate stimuli.

In his first publication Burian mentions that the amount of stimulation exerted by the peripheral images depends on size and brightness of these images and on distance from the fovea. The farther the peripheral stimuli are from the fovea, the larger they must be. Burian, however, does not give experimental evidence for this contention. For this reason I deemed it useful to submit these facts to a further analysis. There was still another factor which seemed of interest: the time of exposure of the peripheral object. During my investigations I had been struck by the fact that under the same experimental conditions and with the same test subject the results differed considerably at different times.

Apparently, there were still many variables. In general, they may be divided as follows: (*a*) Variables derived from a still imperfect method of investigation and apparatus. I shall call them "extrinsic factors." (*b*) Variables derived from factors hitherto unknown, originating in the test subject himself, such as heterophoria, fatigue or work which has been done before starting the experiments. I shall call them "intrinsic factors."

One can reduce the extrinsic factors by a better standardization of the experimental conditions. This was one of the reasons for introducing the time factor: The duration of the peripheral stimuli might be of influence on the result; it might be possible that a very short exposure to the peripheral stimuli might not result in peripheral fusion, whereas a longer exposure might have a positive effect.

PRESENT INVESTIGATION

I had arrived at the concept of peripheral fusion by a method completely different from Burian's. I had been most impressed by the fact that in certain circumstances fusion of images situated on the foveas of the two eyes could be broken up by peripheral stimuli. I therefore used the disturbance of central fusion as a criterion for the existence of peripheral fusion.

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From the Eye Clinic of the University of Amsterdam; Dr. A. Hagedoorn, Director.

1. Burian, H. M.: Fusional Movements: Role of Peripheral Retinal Stimuli, Arch. Ophth. **21**:486 (March) 1939.

Method.—The test is carried out in a special room which can be completely darkened and which can be divided by a drop curtain into two separate parts (fig. 1). One of these parts contains two projection lanterns (Leitz VIII's). The light from these lanterns passes through two small apertures in the drop curtain to the other experimental part of the room. No light can filter from the projection cubicle into the other part except the light from the two projectors. One of the lanterns serves for the projection of two small round dots, intended to be observed haploscopically by the fovea of both eyes; the light from this lantern passes through one of the openings in the drop curtain, then through two Risley rotating double prisms and, finally, through two polaroid® filters. I have described a similar arrangement in a previous paper.[2]

The dots are imaged on an aluminized screen (fig. 1), the dimensions of which are 4 by 4 M. The rotating double prisms are so adjusted that the distance between the images of the two dots is 19 cm., corresponding to an angle of about 2 degrees and 25 minutes when the test subject is seated at a distance of 5 M. from the screen. The polaroid® filters are so rotated that only the eye intended will be stimulated by the dot projected for this eye when the test subject is provided with polaroid® spectacles. In a completely darkened room, these dots, which are imaged haplo-

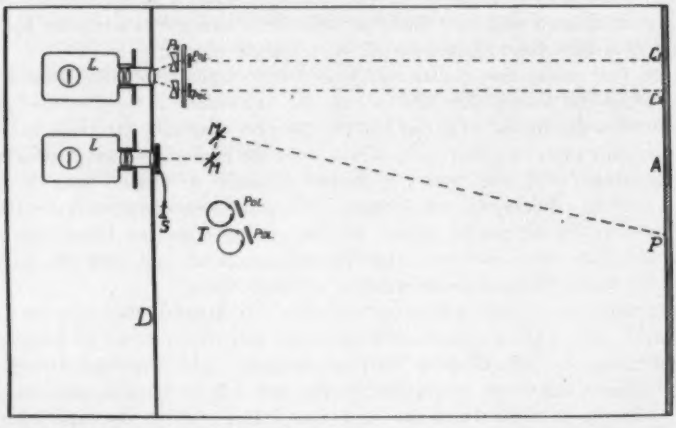

Fig. 1.—Diagram of dark room and projection cubicle. *A*, aluminized screen; *L*, Leitz projectors; *T*, eyes of test subject; *Pol*, polaroid®; *Pr*, prisms; *S*, Compur shutter; *M*, mirrors; C_1 and C_2, central objects; *P*, peripheral object; *D*, drop curtain. The dotted lines indicate the path of the light rays.

scopically on the foveas, are immediately fused by the test subject. The second projector serves for the projection of one peripheral object. The light from this projector passes through the other opening in the drop curtain, which, however, is closed on one side by a Compur shutter; this makes it possible to give time exposures of the peripheral object. The light from this projector then falls on two mirrors, which can be rotated so as to make it possible to project the peripheral object on any desired part of the aluminized screen. The image of the peripheral object is seen by both eyes, as the light from this projector is not intercepted by polaroid® material.

Figure 2 represents a schematic drawing of what actually happens. The left and the right eye are represented by two semicircles. C_1 and C_2 are the central dots which are imaged on the foveas of the left and right eyes, respectively. Another method of observation is also possible,

2. Winkelman, J. E.: Projection Tachystoscope for Orthoptic Treatments: A Preliminary Report, Am. J. Ophth. **33**:461 (March) 1950.

i. e., C_2 is seen by the fovea of the left eye and C_1 by the fovea of the right eye. We called this heteronymous observation, in contradistinction to the method represented by figure 2, which was called homonymous observation. C_1 and C_2 are imaged on corresponding points and are therefore fused to one central dot. Then the peripheral object, P, is exposed, which can be seen with both eyes. P is imaged on the retina of the left eye at P_L and on the right eye at P_r. P_L and P_r are disparate points, the stimulation of which induces a peripheral fusional movement, breaking the fusion of the central dots. As a consequence, C_1 and C_2 are no longer imaged on corresponding points and are now seen by the test subject as two dots. An analogous explanation holds good for the case of heteronymous observation of the central stimuli.

The course adopted in my experiments was as follows: The test subject was placed in a completely darkened room at a distance of 5 M. from the aluminized screen and provided with polaroid® spectacles. The central dots intended for the foveas were projected on the screen so that they were seen either homonymously or heteronymously. The distance between the central dots on the screen corresponded to an angle of 2 degrees and 25 minutes. They were immediately fused into one dot. Then the peripheral object was projected with time exposure

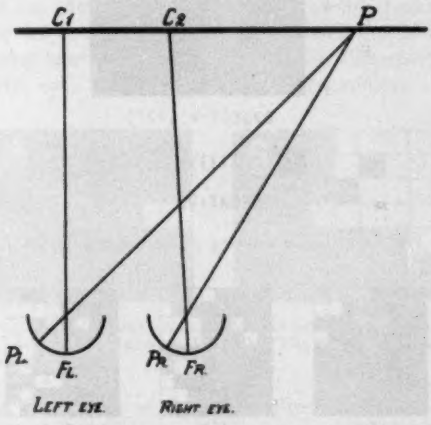

Fig. 2.—Diagram showing stimulation of both retinas of the test subject. C_1 and C_2 are central dots; P is the peripheral object; P_L, image of P on left eye; P_r, image of P on right eye.

at a distance varying from 250 to 20 cm. from the central dots. This corresponded to an angle of from 27 to 2 degrees. The peripheral object was a square of varying dimensions. The edge of the largest peripheral object used corresponded to about 17 degrees (object 1); the other objects had edges ½, ¼, ⅛ and 1/32 the size of the largest square. The smallest object, 1/32, had edges corresponding to ½ degree. The test subject then had to state whether he saw one or two central dots when the peripheral object was suddenly exposed for a short time. In case he saw two central objects, the peripheral fusion was stronger than the central fusion, and the result was noted as positive. The peripheral object was then first projected 27 degrees peripherally, with time exposures of 1, ½, ⅛, 1/10, 1/50, 1/100 and 1/150 second. Then it was projected more centrally, and the same time exposures were made. Positions for the peripheral object were chosen 27, 22, 17, 13, 11, 9, 7, 5 and 2 degrees from the central dots.

The results of these experiments were plotted in graphs as in figure 3. The time of exposure of the peripheral object was recorded on the abscissas; the distance of the peripheral object from the central objects was noted on the ordinates. When the central fusion was broken by the peripheral fusion, the corresponding square was made black, thus indicating a positive result. A blank square means that no influence on central fusion was exerted by the peripheral

stimuli. In figure 4, five of these graphs are given for sizes of the peripheral object of 1 (17 degrees), ½, ¼, ⅛ and 1/32 (½ degree). Each graph represents the experimental results of one session. From these graphs the following conclusions are presented.

OBSERVATIONS

Influence of Exposure Time of Peripheral Object on Peripheral Fusion.—Most of the negative results are tabulated at the right of the graphs; this means that the

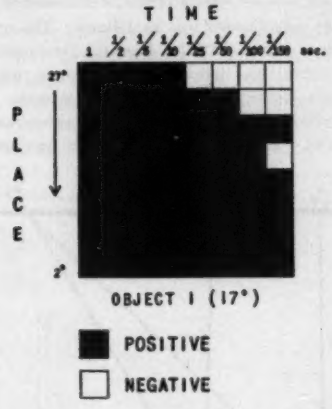

Fig. 3.—Diagram showing method of registration.

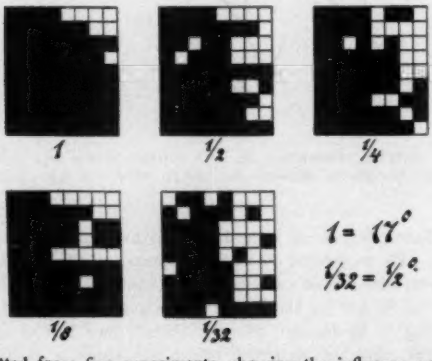

Fig. 4.—Data plotted from five experiments, showing the influence on peripheral fusion of exposure time, distance between fovea and peripheral object and size of peripheral object. The method of registration is the same as that represented in figure 3.

shortest exposures were responsible for most of the negative results. With longer exposures (1 second) peripheral fusion was evident in almost all cases. Most of the negative results can, moreover, be found in the superior part of the graphs: The influence of exposure time becomes evident only when the peripheral object is imaged at a certain distance from the central objects. No absolute liminal time of exposure which can still produce peripheral fusion could be demonstrated by this method.

Influence of Distance Between Fovea and Peripheral Object.—As most of the blank squares are in the superior part of the graphs, it is evident that the intensity of the peripheral fusion decreased toward the periphery.

Influence of Size of Peripheral Object.—The intensity of peripheral fusion increased with a larger peripheral object, especially when the latter was imaged at a greater distance from the fovea. This is clearly demonstrated by comparison of the results obtained with a peripheral object size 1 and those obtained with the smallest peripheral object (1/32).

Influence of Brightness of Peripheral Object.—This was investigated in the following manner: The same method was used as in the foregoing experiments, but in the present test the peripheral object size 1 was exposed with a brightness of either 35 or 3.5 lux. The lower brightness was obtained by putting a Tscherning photometric glass no. 1 before the projection lantern of the peripheral object. Only one tenth of the incident light is transmitted by this glass. During the experiments the test subject did not know whether the peripheral object was projected with a brightness of 35 or 3.5 lux. The series were carried out in one session. The object with the lowest brightness was still of sufficient intensity to be easily perceived by the observer even with the shortest time of exposure. The results of the

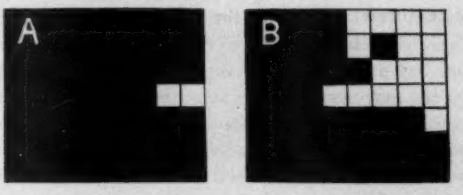

Fig. 5.—Diagram showing influence of brightness of peripheral object on peripheral fusion (*A*, brightness of 35 lux; *B*, brightness of 3.5 lux). The method of registration is the same as that represented in figure 3.

series are given in the graphs of figure 5. From these, it is evident that the object with the higher brightness caused a stronger peripheral fusion than did the other one.

Conclusion.—I find that Burian's contentions are confirmed. In addition, the time of exposure of the peripheral stimulus is found to play a role. In summary, it may be stated that peripheral fusion is strongest under the following conditions: (1) a large peripheral object; (2) a long time of exposure; (3) high brightness of the peripheral object; (4) the peripheral object imaged on the paramacular areas.

From conditions *1*, *2* and *3* it seems possible that the total amount of light of the peripheral stimulus determines the peripheral fusion. From condition *4*, it follows that there is also a qualitative difference between stimuli of the same size, brightness and time of exposure, with respect to whether they are imaged on the paramacular areas or more peripherally.

COMMENT AND SUMMARY

A method is described for the measurement of peripheral fusion. This method differs essentially from that employed by Burian. In my experiments two central

objects are seen haploscopically and one peripheral object is seen with both eyes, whereas in Burian's experiments two peripheral objects were observed haploscopically and often one central object was used, which was fixated by both eyes; otherwise, two central objects were used, which were not fused, but served only to mark the displacement of the eyes. My method is based on the fact that central fusion may be broken up by disparate peripheral stimuli. In addition, I introduced time exposure for the peripheral object. After the test subject has obtained fusion of the two central objects, the peripheral object is exposed for a second or a fraction of a second. This peripheral object, which is seen by both eyes, is imaged on the retinas on disparate points. Stimulation of these peripheral disparate points induces a fusional movement, and consequently central fusion is broken up and the test subject sees two central objects instead of one. This method necessitates a room which can be darkened completely and which is provided with a separate projection cubicle.

With this apparatus I was able to prove that the effect of the peripheral stimuli is influenced by size, brightness, time of exposure and distance from the fovea of the peripheral object. From the first three conditions it seems probable that the total quantity of light determines the strength of the peripheral fusion. From the last, it is apparent that with regard to the effect of peripheral stimuli there is a qualitative difference between the different zones of the retinal periphery.

EXPERIMENTAL STUDIES ON EARLY LENS CHANGES AFTER ROENTGEN IRRADIATION

II. Exchange and Penetration of Radioactive Indicators (Na^{24}, K^{42}, I^{131}, P^{32}) in Normal and Irradiated Lenses of Rabbits

LUDWIG VON SALLMANN, M.D.
AND
BEATRICE D. LOCKE, M.A.
NEW YORK

RADIOACTIVE tracers were employed in this part of the study to investigate the effect of roentgen irradiation on the permeability of the lens and of the blood-aqueous barrier as far as it could be deduced from this order of experiment. According to many workers, changes in the permeability of phase boundaries may play an important part in the pathogenesis of roentgen ray cataract. It is widely held, moreover, that the biologic actions of ionizing radiations are based partly or entirely on alterations in the selective permeability of the cell membrane. These changes can influence the rate of entrance of substances into the cells, and of their elimination, and thus may modify metabolic and synthetic processes. To the best of our knowledge, no extensive investigations on the eye which could lend support to this theory or disprove it had been carried out at the time the present study was undertaken.

Two experimental technics, each in part supplementary to the other, have been followed. The specific activities of the labeled substances have been determined in body fluids and digests of the lens (1) by means of the Geiger-Müller counter or (2) by an autoradiographic procedure which, when combined with a densitometric method, has also allowed quantitative estimations to be made.

EXPERIMENTAL PROCEDURES

In general, the right eye of each of a group of young, mature chinchilla rabbits (57 experiments) was irradiated with 2,000 or 1,500 r. The radiation factors were the same as in the first part of the study (220 kv., 20 ma., 0.5 mm. Cu + 1 mm. Al filtration, at 30 cm. target skin distance, 20 mm. field (round), half-value layer 1 mm. Cu, 145 r per minute in air. The left eye was shielded and outside the beam. The amount of indirect irradiation to which the left eye may have been exposed during the treatment of the right eye was measured in individual experiments by means of an ionization chamber, which was placed in the left socket after enucleation. Clinical changes were recorded when they occurred before the experiment was

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terminated. Radioactive material was introduced by the intraperitoneal route at stated intervals prior to enucleation. The directly irradiated and the control eyes were removed at periods varying from two to eight weeks after irradiation, or later when cataractous changes were advanced.

The radioactive sodium and radioactive potassium were produced in the cyclotron at Columbia University and delivered to the laboratory in dilute solutions of sodium chloride and potassium chloride, respectively. Radioactive inorganic iodine was supplied by the Department of Radiological Research at the College of Physicians and Surgeons in the form of a solution of the carrier-free sodium salt. Radiophosphorus, supplied by the same source, was used in the form of phosphoric acid. Serum levels were determined on samples of heart blood obtained immediately before withdrawal of 0.1 cc. of aqueous humor from each eye. Micropipets insured the accurate measurement of these small quantities. The lenses were removed carefully after circumcorneal or equatorial dissection of the globes and were freed on moist filter paper from adherent uveal pigment and strands of vitreous. They were then transferred directly to flat tin pans for weighing and heat digesting with 5 cc. of 5 per cent sodium hydroxide. The activity of the fluid samples was determined in the usual way with the Geiger-Müller counter. The aqueous humor and blood serum were usually diluted to 10 and 25 cc., respectively, with distilled water and measured in aliquots in the tin pans. In other experiments the eyes were removed at desired intervals after injection of the radioactive substances, quickly frozen in freezing mixtures and dissected with chilled microtome knives to prepare slices 1.5 to 2.0 mm. thick adjacent to the vertical meridian. In these experiments great care was exercised to obtain slices of equal thickness from the two eyes and to assure identical photographic treatment of the preparations.

The technic applied for obtaining autoradiograms of the frozen slices was described in an earlier paper.[1] The densities of the films in various areas of the photographic imprint were measured with a sensitive photometric device, as previously described.[2] Exposure and development times were varied according to the amount of the radioactive element expected in the preparations and according to the energy of the radiations of the tracer. Modifications of the technic in connection with the use of individual radioactive isotopes will be discussed in the sections describing the results.

RESULTS

Experiments with Radiosodium (34 experiments).—The largest group of experiments was carried out with radiosodium (34 animals). The amount of the injected labeled indicator, expressed in terms of radioactivity, varied from 0.6 to 0.9 millicurie per kilogram of body weight. Specific activities in blood serum, aqueous humor and lens digests of both eyes were measured in nonirradiated animals to determine the magnitude of error (five experiments) occurring with the technic employed in this study and in rabbits given radiation of 2,000 r to one eye. These animals were killed at periods of two weeks (six experiments), four weeks (eight experiments), six weeks (eight experiments) and eight weeks (one experiment) after the irradiation. As was shown by the studies of Kinsey and co-workers,[3] equilibrium between the activity levels for plasma and aqueous humor is almost reached four hours after intraperitoneal introduction of the tracer. The passage of the radioactive compound across the blood-aqueous barrier was tested, therefore, at intervals of one and two hours after injection. Five experiments were terminated

1. von Sallmann, L.; Evans, T. C., and Dillon, B.: Studies of the Eye with Radiosodium Autographs, Arch. Ophth. **41**:611-626 (May) 1949.

2. von Sallmann, L., and Dillon, B.: Studies of the Eye with Radioiodine Autographs, Am. J. Ophth. **33**:429-440, 1950.

3. Kinsey, V. E.; Grant, W. M.; Cogan, D. C.; Livingood, J. J., and Curtis, B. R.: Sodium, Chloride and Phosphorus Movement and the Eye Determined by Radioactive Isotopes, Arch. Ophth. **27**:1126-1131 (June) 1942.

four hours after the use of the tracer. Measurements with the Geiger-Müller counter were also taken on three animals with various stages of roentgen radiation cataract. The results were compared with observations on autoradiographs from normal eyes and irradiated clear lenses and with densitometric readings on autographs obtained from eyes with advanced lenticular opacities caused by roentgen radiation.

The comparison of measurements on the aqueous humor and the lens from the right and left eyes of five normal, nonirradiated animals showed good agreement, as the differences for the lenses did not exceed 9 per cent and those for the samples of aqueous humor 3 per cent (table 1). When one eye was exposed to 2,000 r, the contralateral orbit received an amount of 7 r by indirect irradiation.

The exchange of sodium in the control lenses with the systemically introduced labeled element was small at the early intervals of one and two hours, and the specific activities of the digests were of such low order that surface contamination

TABLE 1.—*Radioactivity Measurements* of Aqueous Humor and Lens from Right and Left Eyes of Nonirradiated Rabbits Two Hours After Systemic Administration of Radiosodium*

Rabbit No.		Aqueous	% Difference	Lens	% Difference	Serum
1	O. D.	10,580	3.0	179	6.1	.....
	O. S.	10,200	...	190	...	.....
2	O. D.	8,976	1.8	...	...	10,718
	O. S.	8,822	...	179	...	.....
3	O. D.	12,006	0.4	291	4.4	18,105
	O. S.	12,911	...	304	...	.....
4	O. D.	14,060	2.2	275	0.0	20,006
	O. S.	14,350	...	275	...	.....
5	O. D.	12,000	1.0	199	9.0	22,610
	O. S.	12,120	...	217	...	.....

* Geiger-Müller counts per minute per cubic centimeter or per gram.

of the preparations with traces of aqueous humor and vitreous fluid may have influenced the results. Analogous observations were made on autographs of normal or not directly irradiated lenses; i. e., with the technic used the photographic effects of such lenses were minimal when they were compared with the background readings.

In lenses of directly irradiated eyes the uptake of the labeled sodium seemed increased two weeks after exposure of the eye to 2,000 r. The radioactivity of the digest was 10 per cent greater than that of the controls when one hour had elapsed after the injection of the indicator and was 8 per cent greater at the two hour interval. However, the specific activities of samples of aqueous humor also exceeded those of the control eyes by 24 per cent one hour after injection and by 8 per cent at the two hour period. Four weeks after irradiation the average difference between the experimental and the control eyes in the one and two hour experiments was 22 and 9 per cent for the lenses and 21 and 9 per cent for the aqueous humor. At the six week period the percentage increases in specific activity of the irradiated over the control lenses were 52 and 35 in the determinations one and two hours after injection. Samples of aqueous humor of these eyes showed smaller deviations except in one experiment. In this group peripheral cataractous changes were noted clinically or on dissection of the globes, but the irradiated and control lenses of individual

animals did not vary significantly in weight. Table 2 indicates the disparity of the results in single experiments.

TABLE 2.—*Radioactivity Measurements* on Aqueous Humor and Lens from Irradiated† and Control Rabbit Eyes After Systemic Administration of Radiosodium‡*

Rabbit No. Experiment	Aqueous	% Increase	Lens	% Increase	Serum
Two Weeks After Irradiation					
A 1 hr. after intraperitoneal injection of Na^{24}					
1 O. D.	3,300	18	41	0	7,240
O. S.	2,800	..	41	..	.....
2 O. D.	15,200	28	185	20	22,650
O. S.	12,000	..	154	..	.....
3 O. D.	4,320	28	64	10	10,954
O. S.	3,380	..	48	..	.....
B 2 hr. after intraperitoneal injection of Na^{24}					
4 O. D.	8,180	14	143	13	12,580
O. S.	7,200	..	125	..	.....
5 O. D.	12,720	1	222	2	19,490
O. S.	12,580	..	217	..	.....
C 4 hr. after intraperitoneal injection of Na^{24}					
6 O. D.	26,000	7	512	8	.....
O. S.	24,220	..	474	..	.....
Four Weeks After Irradiation					
A 1 hr. after intraperitoneal injection of Na^{24}					
7 O. D.	3,450	11	53	20	7,765
O. S.	3,100	..	44	..	.....
8 O. D.	11,280	33	150	38	25,235
O. S.	8,460	..	108	..	.....
9 O. D.	7,150	20	111	9	23,495
O. S.	5,950	..	102	..	.....
B 2 hr. after intraperitoneal injection of Na^{24}					
10 O. D.	14,140	13	351	17	18,840
O. S.	12,560	..	300	..	.....
11 O. D.	9,420	5	115	0	10,855
O. S.	8,900	..	115	..	.....
C 4 hr. after intraperitoneal injection of Na^{24}					
12 O. D.	35,600	18	812	23	.....
O. S.	30,100	..	624	..	.....
13 O. D.	76,780	0	1,576	35	.....
O. S.	77,080	..	1,164	..	.....
14 O. D.§	45,250	5	1,642	79	.....
O. S.	43,300	..	912	..	.....
Six Weeks After Irradiation					
A 1 hr. after intraperitoneal injection of Na^{24}					
15 O. D.	10,600	24	145	67	27,168
O. S.	8,620	..	87	..	.....
16 O. D.	11,560	33	202	49	14,610
O. S.	8,700	..	135	..	.....
17 O. D.	9,070	26	133	33	12,360
O. S.	7,180	..	100	..	.....
18 O. D.	2,610	0	48	60	4,418
O. S.	2,750	..	30	..	.....
B 2 hr. after intraperitoneal injection of Na^{24}					
19 O. D.	9,280	28	270	23	12,065
O. S.	7,240	..	220	..	.....
20 O. D.	12,720	32	248	44	16,155
O. S.	9,660	..	172	..	.....
21 O. D.	12,530	29	329	29	14,320
O. S.	9,640	..	256	..	.....
22 O. D.	9,700	11	260	44	12,310
O. S.	8,740	..	181	..	.....

* Measurements in Geiger-Müller counts per minute per cubic centimeter or gram.
† The right eye was exposed to 2,000 r in each set of experiments.
‡ Dose of radiosodium was 0.6 to 0.9 millicurie per kilogram of body weight.
§ Corneal burn and conjunctival discharge.

Measurements of the radioactivity of lenses with advanced cataractous changes were obtained in three experiments but could be compared with counts for clear lenses in control eyes in two experiments only. One hour after injection of the

labeled compound the activity of the digest from a lens with total roentgen ray cataract was about 20 times as great as the value obtained for the normal, control lens, whereas samples of aqueous humor from the two eyes gave almost equal counts. A partially cataractous lens had an activity 44 per cent higher than the clear lens of the other eye. In a third experiment the lens of the right eye, with extensive opacities in the anterior and posterior cortices, was compared with an intumescent cataract of the left eye. Both eyes had been irradiated. Although the counts were low because of the moderate level of activity in the serum of this animal, they were three times as high in the swollen, left lens as in the right lens (table 3).

The existence of a relationship between the stage of cataract formation and the specific activity of the lens digest was confirmed by a comparison of the autographs of eyes with roentgen ray cataract with autographs of the control eyes. The exami-

TABLE 3.—*Radioactivity Measurements on Aqueous and Lens of Irradiated Rabbit Eyes with Advanced Cataractous Changes and of Control Eyes After Systemic Administration of Radiosodium* *

A. Geiger-Müller Counts per Min. per Cc. or Gm.

Rabbit	Eye	Aqueous	Lens	Time After Injection
30	Partial cataract	.....	772	4 hr.
	Normal lens	.....	535	
34	Total cataract	7,900	1,817	1 hr.
	Normal lens	8,120	90	Serum: 13,110 c./m.†
35	Partial cataract	1,800	23	1 hr.
	Intumescent cataract	2,400	60	Serum: 5,690 c./m.

B. Densitometric Readings of Radioautographs ‡

Rabbit	Eye	Anterior Chamber	Lens	Time After Injection
31	Partial cataract	0.18	0.05	4 hr.
	Normal lens	0.13	0.02	
32	Advanced cataract	0.18	0.14	4 hr.
	Normal lens	0.17	0.01	
33	Advanced cataract	0.14	0.37	4 hr. Intravitreal
	Normal lens	0.12	0.15	injection of 50 μc.

* Dose of radiosodium was 0.6 to 0.9 millicurie per kilogram of body weight.
† Counts per minute.
‡ Background of films 0.00 density.

nation with the densitometer indicated that the photographic effect of the cataractous lens was 14 times as great as that produced by the control lens. The darkening of the film in the area of the opaque lens was diffuse in character when the experiment was terminated four hours after the injection of the labeled compound. In an early stage of a partial radiation cataract the photographic imprint of the lens did not differ noticeably from that of the clear lens. In one animal the radiotracer was injected into the vitreous of both eyes in equal amounts (50 microcuries). The autograph showed the uptake of Na^{24} from the cataractous lens to be significantly greater than the uptake from the clear lens of the other eye.

Experiments with Radiopotassium (8 experiments).—Radioactive potassium was injected in the form of potassium chloride. The activity of the introduced material was estimated to be 300 microcuries per kilogram of body weight. Since no data were available on the turnover of this indicator in the rabbit eye, determinations were carried out on 11 nonirradiated eyes one, four and 18 hours after the intraperitoneal use of the labeled compound. The autographic method was used with six of these eyes. Moreover, the distribution of the tracer was studied

in two eyes with partial radiation cataract and in three eyes in which the radiation damage had led to complete opacification of the lens.

One hour after the injection of K^{42} the individual activities in samples of blood serum and in fluids and tissues from the normal eye descended in the following sequence: extraocular muscles, anterior uvea, cornea, serum, aqueous humor, lens and vitreous. One hour after the injection digests of the lens contained 59 per cent of the amount determined in the aqueous humor and about four times the amount measured in the vitreous fluid. At the four hour interval the lens exhibited 91 per cent of the activity of the aqueous humor and approximately 2.5 times that of the vitreous humor. Autographs of this stage provided additional information on the exchange of K^{42} with the potassium in the lens. The photographic effect of radiopotassium in the anterior cortex and in the region of the equator exceeded that of the indicator in the aqueous humor and in the cornea. From the densitometric read-

TABLE 4.—*Radioactivity Measurements on Fluids and Tissues from Nonirradiated and Irradiated Rabbit Eyes After Systemic Administration of Radiopotassium* *

	Geiger-Müller Counts per Min. per Cc. or Gm.								Time After Injection, Hr.
	Aqueous	Lens	Cornea	Serum	Vitreous	Sclera	Iris-Ciliary Body	Muscle	
Normal lens.....	870	440	1,363	1,586	111	.....	5,123	5,350	1
Normal lens.....	1,180	702	1,588	1,442	295	.....	5,615	6,940	1
Normal lens.....	1,170	706	1,610	1,442	315	.....	4,511	6,940	1
Normal lens.....	2,330	2,125	3,170	2,860	860	2,961	6,767	.....	4
Partial cataract.....	2,170	1,926	3,500	2,860	800	.....	7,724	14,750	4
Partial cataract.....	2,020	1,502	3,680	2,224	867	2,165	8,460	8,400	4
Total cataract.....	2,330	1,430	4,250	2,234	847	3,000	8,880	.....	4
Normal lens.....	1,760	5,172	2,798	1,714	...	.....	.....	.....	18

Average Densitometric Readings on Radioautographs of Frozen Slices of Nonirradiated Rabbit Eyes

No. of Eyes	Time After Injection, Hr.	Anterior Chamber	Lens Area †			
			Nucleus	Anterior Cortex	Equator	Posterior Cortex
1.....	1	0.03	0.01	0.035	0.045	0.005
4.....	4	0.11	0.09	0.15	0.16	0.09
1.....	18	0.12	0.22	0.21	0.22	0.19

* Approximate dose 0.3 millicurie per kilogram of body weight.
† Background of film 0.00 density.

ings it could be seen that four hours after injection the accumulation of the labeled element in the mentioned parts of the lens was about 85 per cent as great as in the nucleus of the lens and in the posterior cortex (table 4). In measurements carried out 18 hours after injection of radiopotassium the lens contained the indicator in an amount about three times as great as that measured in serum and aqueous humor per weight unit and about twice the amount recorded in the cornea. Autographs of this stage demonstrated an almost homogenous distribution of the tracer in the lens.

The comparison of the activities in tissue obtained from control eyes and from irradiated eyes with initial lenticular opacities indicated a slightly smaller uptake of K^{42} in the lens with the radiation damage. In an experiment concluded four hours after injection of radiopotassium the lens with total cataract of the right eye contained 11 per cent less of the indicator than did the partial cataractous lens of the left eye. On the other hand, the radioactivities of samples of aqueous humor and cornea from the right eye, were 15 per cent higher than the activities of samples from the left eye.

It was obvious from the results secured with two methods of measuring radioactivity that the uptake of K^{42} by the lens from the aqueous humor surpassed markedly the uptake of radiosodium in normal eyes. Autographs taken at various periods after the introduction of the indicator demonstrated the movement of radiopotassium from the surrounding ocular fluids across the anterior surface of the lens and the retention in the lens of a concentration which surpassed the concentrations present in other tissues and fluids of the eye. Whereas the uptake of radiosodium in lenses with total radiation cataract was increased about 20 times that in the control eyes, the amount of radiopotassium in cataractous lenses was slightly decreased.

TABLE 5.—*Radioactivity Measurements on Aqueous Humor and Lens from Nonirradiated and Irradiated Rabbit Eyes Twenty-Four Hours After Systemic Administration of Radioiodine* *

		Geiger-Müller Counts per Min. per Cc. or Gm.								
Rabbit Eye		Aqueous	Lens	Serum	Rabbit Eye		Aqueous	Increase	Lens	Increase	Serum
A	Normal lens	1,330	82	.....	116	Partial cataract	1,090	4%	111	2×	.....
	Normal lens	1,250	67	2,786		Normal lens	1,020	....	51	....	2,760
B	Normal lens	2,430	161	.....	121	Partial cataract	2,020	12%	116	71%	.....
	Normal lens	2,280	111	5,178		Normal lens	1,800	....	68	....	3,850
174	At 2 wk.	580	44	.....	29	Total cataract	1,940	2×	1,187	20×	.....
	Normal lens	520	25	1,240		Normal lens	900	....	57	....	3,990
173	At 2 wk.	200	25	.....	38	Total cataract	3,140	2×	1,605	11×	.....
	Normal lens	200	25	580		Partial cataract	1,780	....	147	....	3,400
130	At 3 wk.	960	84	.....	32	Total cataract	4,100	28%	1,584	14×	.....
	Normal lens	920	80	5,470		Normal lens	3,205	....	110	....	.....
50	Initial cataract	2,590	97	.....	4	Total cataract	.....	....	2,016	....	.....
	Normal lens	2,080	66	.....		Total cataract	1,207	....	2,094	....	.....
109	Initial cataract	450	72	.....							
	Normal lens	450	48	2,810							

Average Densitometric Readings from Radioautographs of Frozen Slices of Similar Eyes

Rabbit Eye		Anterior Chamber	Lens	Rabbit Eye		Anterior Chamber	Lens
21	Initial cataract	0.073	0.006	52	Total cataract	0.09	0.19
	Normal lens	0.075	0.006		Normal lens	0.09	0.008
154	Partial cataract	0.08	0.06	44†	Partial cataract	0.025	0.14
	Normal lens	0.025	0.008		Normal lens	0.05	0.06

* Dose of radioiodine was 1 millicurie per kilogram of body weight.
† Four hours after intravitreal injection of 40 microcuries.

Experiments with Radioiodine (17 experiments).—In this series of experiments radioactive inorganic iodine was injected in a dose with an activity of about 1 millicurie per kilogram of body weight. The examinations were performed 24 hours after injection.

Digests of normal lenses and of control lenses of irradiated animals gave low counts and thus corroborated the observations of minimal photographic effects on lenses shown autographically in a previous study in which a similar dose was used.[2] The penetration of radioiodine in the control lenses equaled the uptake in the lenses of irradiated eyes before lenticular opacities had developed. With the appearance of cataractous changes more of the indicator passed into the lens. The activities of the digests of such lenses 24 hours after injection were from 50 to 100 per cent greater than those of the control eyes. Autographs also illustrated these differences satisfactorily. With this technic the higher uptake by a lens with early cataractous changes was also observed after injection of the labeled compound into the vitreous. Measurements on digests of lenses (table 5) with total cataract gave high readings,

ranging from 15 to 20 times as great as those on the controls. The darkening of the film by such lenses expressed in densitometric units also surpassed about 20 times the readings in the area of the lens of control eyes.

At the 24 hour period there was close analogy between the activity values for the blood serum and those for the aqueous humor. Measurements on samples of aqueous humor gave similar results for normal and for irradiated eyes three weeks after exposure to 2,000 r and 11 to 12 weeks after irradiation with 1,500 r. In contrast to these observations were those on eyes with complete radiation cataract in which 24 hours after injection of I^{131} a substantial increase in specific activity was measured in the aqueous humor of the irradiated eye. The increments amounted to from 28 to 100 per cent over the readings for the control eyes. In an experiment in which both eyes had been irradiated but the cataractous process had reached a stage of complete opacification only in the right eye, the activity of the aqueous humor in this eye showed twice the value for the left eye, with partial opacification of the lens. In this experiment counts on digests of the lenses were 12 times as

TABLE 6.—*Radioactivity of Fractionated Lens Digests Twenty-Four Hours After Systemic Administration of Radioiodine* *

Rabbit	Eye	Geiger-Müller Counts per Min. per Gram: Acid-Soluble Nonprotein Fraction	Geiger-Müller Counts per Min. per Gram: Lipid-Protein Fraction
50	Initial cataract	98	4
	Normal lens	52	14
32	Total cataract	1490	44
	Normal lens	44	16
4	Total cataract	1944	72
	Total cataract	2022	74

* Dose of radioiodine was 1 millicurie per kilogram of body weight.

high in the eye with the total radiation cataract. In two experiments the specific activities of cataractous lenses were per weight unit higher than the activities of the aqueous humor 24 hours after injection. In two similar examinations, however, this relationship appeared reversed. The differences in radioactivity between cataractous and control lenses always exceeded greatly the differences between samples of the aqueous humor from such eyes.

By means of the technic described in an earlier paper [2] the content of I^{131} in the acid-soluble fraction in a lens with full radiation cataract was compared with the activity of the protein lipid residue. Radioactivity was demonstrated almost exclusively in the acid-soluble part, suggesting the accumulation of the radioindicator in the cataractous lens as the inorganic salt. A scarcely perceptible amount of specific activity was measured in the protein lipid residue. This may indicate that a minute amount of radioiodine was bound organically to the lens proteins.

Experiments with Radiophosphorus (15 experiments).—In this series of experiments the eyes were removed 24, 48 or 96 hours after the introduction of 0.8 to 1 millicurie of labeled phosphorus per kilogram of body weight. The distribution of the indicator was studied by the photographic method only. It is evident from the reproduced autographs of normal eyes that P^{32} was preferentially

exchanged in the equatorial part of the normal lens. The darkening of the film corresponded first to the superficial layers of this part of the lens and subsequently also to the deeper parts. As shown by the shading of the film emulsion, the labeled phosphate later accumulated similarly in the superficial layers of the anterior and posterior cortices, with the amount decreasing toward the supernuclear zone. Densitometric examination of the autographs showed the percentage distribution.

When radiation injury to the lens was in its initial stage, this pattern of the P^{32} distribution showed no appreciable changes. In lenses with full radiation cataract, however, the photographic representations contrasted sharply with those of normal lenses and showed clearly that the exchange with the introduced radiophosphorus in the lens cortex was almost abolished, only two dark lines on the lens surface behind the equator expressing activity in a thin, superficial zone.

TABLE 7.—*Densitometric Readings on Radioautographs of Irradiated and Control Eyes After Systemic Administration of Radioactive Phosphorus* *

Rabbit	Eye	Anterior Chamber	Lens Area †				Time After Injection
			Nucleus	Anterior Cortex	Equator	Posterior Cortex	
17	3 wk. after 1,500 r	0.10	0.00	0.00	0.22	0.04	24 hr.
	Normal lens	0.09	0.00	0.09	0.22	0.04	
22	4 wk. after 1,500 r	0.05	0.05	0.09	0.16	0.09	72 hr.
	Normal lens	0.06	0.04	0.08	0.16	0.07	
15	Initial cataract	0.10	0.10	0.17	0.24	0.14	5 days
	Normal lens	0.09	0.10	0.16	0.25	0.14	
11	Advanced cataract	0.07	0.08	0.06	0.09	0.08	4 days
	Normal lens	0.07	0.07	0.11	0.20	0.09	
25	Advanced cataract	0.16	0.04	0.09	0.12	0.04	4 days
	Normal lens	0.14	0.12	0.20	0.37	0.18	

* Dose of radiophosphorus was 0.8 to 1.0 millicurie per kilogram of body weight.
† Background of film 0.00 density.

COMMENT

The effect of the irradiation with 2,000 r on the blood-aqueous barrier was studied with radiosodium before the exchange equilibrium of the indicator in plasma and aqueous humor had been established. At time intervals of one and two hours after intraperitoneal introduction, more Na^{24} had accumulated in the aqueous humor of the irradiated eye than in that of the control eye in experiments in which there had been a lapse of two, four and six weeks after exposure to roentgen rays. The difference ranged up to 33 per cent. The change in the first four weeks may have been due not only to a direct effect on the barrier membranes, especially on the capillaries of the ciliary processes, but also, in an indirect way, to radiation damage to the cornea and other tissues. At the six week interval, however, when the difference between the two eyes was greatest, the irradiated eyes were white, and acute signs of the corneal lesion had subsided.

In an earlier experimental series on rabbits, the permeability of the barrier for fluorescein was examined in vivo after irradiation of one eye with 100, 250, 500, 1,000 or 2,500 r doses of penetrating roentgen rays.[4] When doses of 500 and 1,000 r were used, the appearance curve of the dye in the aqueous humor reached higher peaks than were reached in the control eyes, but the disturbance lasted only

4. von Sallmann, L., and Dillon, B.: Zur Frage der Strahleneinwirkung radioaktiver Indikatoren in physiologischen Studien am Auge, Wien. klin. Wchnschr. **61**:714-716, 1949.

one week. Irradiation with 2,500 r enhanced the passage of fluorescein, Evans blue (T 1824) and serum proteins for at least 38 days, i. e., for about the same length of time that the passage of radiosodium was enhanced after the use of 2,000 r. The similarity of the results recorded with such different substances suggested that the increment of the Na^{24} values for the aqueous humor after irradiation were due in the main to a nonselective increase of permeability of the ciliary capillaries through which sodium passes with ease. The development of lenticular opacities in the rabbit eye after exposure to 1,000 r or less has been described by various workers and confirmed by unpublished data obtained in this laboratory.[5] The transient nature of the demonstrable alterations of barrier qualities under these conditions makes it unlikely that they constitute a prerequisite to the formation of roentgen ray cataract. Prolonged and severer increases in the permeability of the barrier for blood constituents, caused by noxae of various kinds, usually does not lead to lenticular opacities in rabbits.

The uptake of radioactive indicators was determined by measurements of specific activities with the Geiger-Müller counter on digests of the lens and on samples of aqueous humor and by an autographic technic. The pattern of distribution of the tracers was studied by a densitometric procedure on autographs. It is obvious that the over-all uptake and percentage distribution of the labeled elements in the lens are influenced by factors other than changes in permeability of lens tissue and alterations in the mechanism of active transport at phase boundaries, and that these factors must be considered separately for each individual labeled indicator.

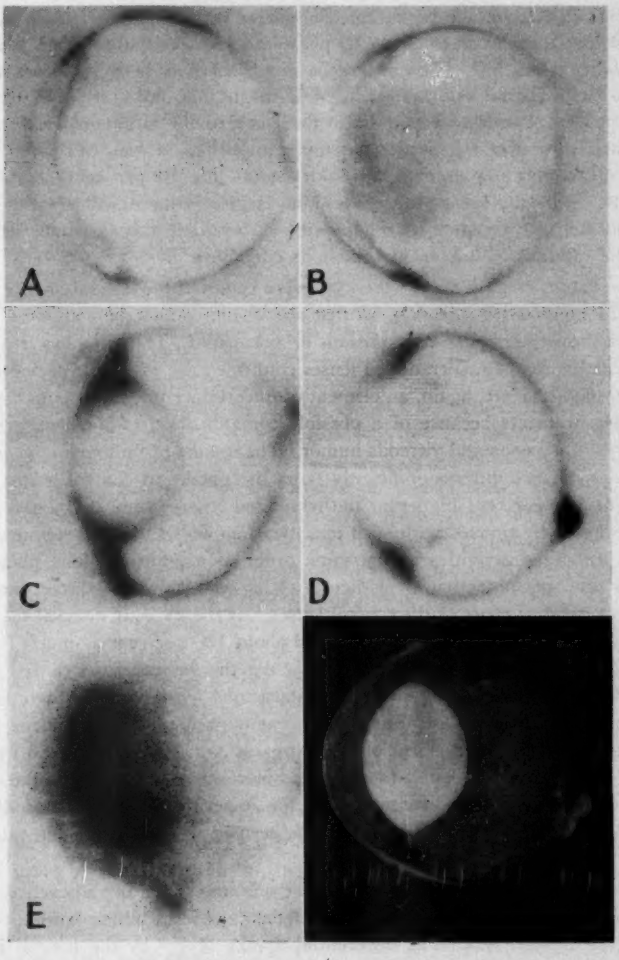
The rate of penetration of Na^{24} into the normal lens was slow, in contrast to the rapid rate of exchange of introduced labeled sodium with the sodium in extracellular fluids. However, with the technic employed, and in view of the relatively short half-life of Na^{24}, it was impossible to follow closely the speed of penetration of minute quantities into the normal lens. Unavoidable contamination of the lens surface with traces of surrounding fluids further tended to invalidate the accuracy of low readings in the Geiger-Müller counter. After irradiation with 2,000 r a moderate increase in the uptake of Na^{24} by the lens ran roughly parallel to the increase of the indicator in the aqueous humor until changes in the transparency of the lens became visible. The increments of radioactivity in the lens in the first four weeks after irradiation did not express, therefore, an increase in the permeability of the tissue for the labeled element.

Initial lenticular opacities are caused to a great extent by the formation of vacuoles and by an accumulation of fluid in extracellular cystic spaces.[6] In the presence of such opacities, more radiosodium was accumulated in the lens than can be explained by the increments of radioactivity in the aqueous humor. The increase of extracellular space in the lens during the progress of cataractous changes, coupled with an increased rate of exchange of the sodium of the lens with the introduced radiosodium, conveyed the impression of a close relationship of the two phenomena. It remained unknown whether or not an increased permeability of the surface layers of the lens for the labeled sodium took part in the greater uptake of the tracer by the cataractous lens.

5. Cataractous changes were observed after a single irradiation with 750 r.

6. von Sallmann, L.: Experimental Studies on Early Lens Changes After Roentgen Ray Irradiation: I. Morphological and Cytochemical Changes, Arch. Ophth. **45**:119-161 (Feb.) 1951.

In contrast to the slow rate of passage of radiosodium from the aqueous humor into the lens, there was a rapid exchange of the systemically introduced radiopotassium and the potassium of the lens. One hour after intraperitoneal injection of the indicator the photographic effect of the anterior cortex and the equator



Radioautographs of rabbit eyes after intraperitoneal injection of radioactive tracers: I^{131}: (*A*) control lens, (*B*) lens with roentgen radiation cataract; P^{32}: (*C*) control lens, (*D*) lens with roentgen radiation cataract; K^{42}: (*E*) normal eye. Frozen eye slice, 1.5 mm. thick.

equaled the effect of the aqueous humor. The ratio of specific activities of the anterior part of the lens cortex and the aqueous humor was considerably above 1 at the four hour interval. In autographs from slices of eyes removed four hours

after the introduction of K^{42}, the area of the ciliary body alone showed more darkening than the anterior cortex of the lens. Determinations of the activities on lens digests at the one hour and four hour periods gave lower counts than those on the aqueous because of the very moderate concentration of K^{42} in the nucleus and in the posterior cortex at this time.

Analytical data on the potassium content of normal and of cataractous lenses indicated that the lens loses 50 to 80 per cent of its potassium when the stage of total cataract is reached.[7] The difference in radioactivity between lenses with total and those with partial cataract was of low magnitude, however. The explanation of this apparent discrepancy may lie in the fact that the target material, potassium metaborate, showed a 0.5 per cent sodium impurity. It was calculated that this amount of sodium contamination would account for 10 per cent of the emitted radiations. Since the uptake of radiosodium is greatly increased when the cataractous process is advanced, it was impossible to evaluate the extent of decrease of K^{42} in the cataractous lens by the methods employed.

In the experiments with I^{131} the eyes were removed for measurements of radioactivity 24 hours after injection, so that the results cannot be compared with the results of the experiments with sodium or potassium. The very low readings on activities in digests of nonirradiated lenses confirmed the results of previous studies with autographs. Here, again, an element of uncertainty prevailed as to the validity of the measurements because of a possible contamination of the lens preparation with traces of aqueous and vitreous humor. The uptake of radioiodine in irradiated clear lenses did not differ significantly from the uptake in control lenses but was from 50 to 100 per cent higher in the irradiated lenses when peripheral opacities had developed. In animals with such opacities samples of aqueous humor from the two eyes exhibited activities of the same order of magnitude. The photographic effect and the specific activities of digests of lenses with total cataract were about 20 times as great as those of the control lenses. At the 24 hour interval the aqueous humor in eyes with total cataract contained about 100 per cent as much I^{131} as the aqueous humor in control eyes. Whether or not this increase was caused by backward diffusion of radioiodine from the cataractous lens, with its high uptake of the tracer, remained in doubt. Since there was experimental proof of the presence in such lenses of I^{131} in the form of the inorganic salt, i. e., in the main not bound to lens protein, it was reasonable to assume an increased permeability of the damaged lens for this radioactive indicator. As in the experiments with radiosodium, the change in rate of penetration of radioiodine seemed to accompany the cataractous process, rather than to precede it.

It could be demonstrated by the systemic administration of radioactive isotopes that phosphorus and potassium, which are elements of intracellular nature, penetrate well into the normal lens. The differential uptake of P^{32} by the normal lens was shown in autographs by the pronounced photographic effect of the indicator at

7. Lebensohn, J. E.: Biochemistry of Lens: Mineral Metabolism in Normal and in Cataractous Lens, Arch. Ophth. **15**:217-221 (Feb.) 1936. Mackay, G.; Stewart, C. P., and Robertson, J. D.: A Note on the Inorganic Constituents of Normal and Cataractous Human Crystalline Lenses, Brit. J. Ophth. **16**:193-201, 1932. Salit, P. W.: Der Mineralbestand der kataraktösen und sklerosierten menschlichen Linsen. Arch. f. Ophth. **139**:654-667, 1938.

the equator of the lens and, later, in the superficial cortical layers, both anteriorly and posteriorly. In general, the accumulation of radiophosphorus and the speed of its exchange with tissue phosphorus are dependent on the rate of metabolism of the tissue. *Ceteris paribus,* rapidly metabolizing tissue shows enhanced incorporation of P^{32}. The growing part of the lens presented by the region of the equator and the surface portion of the cortex can be considered as the seat of a higher metabolic turnover and of processes of rapid phosphorylation. An analytical determination reported by Palm [8] and confirmed in Dr. Dische's laboratory [9] showed a ratio of 1 to 10 in the content of adenosine triphosphate of the superficial part of the cortex of the lens and that of the nuclear part, but no significant differences in the inorganic phosphorus between the two parts.

Initial cataractous changes did not alter the autographic pattern for P^{32}. The steep gradient of densities, decreasing from the surface toward the nucleus of the lens, persisted as long as the main part of the lens remained clear. Thus, neither in clear irradiated lenses nor in those with peripheral opacities did the technic employed demonstrate a depression in the uptake of radiophosphorus, such as was observed early after irradiation of tissue with high mitotic activity—for example, rat liver and sarcoma [10] and lymphomatous tissue in mice.[11] On the other hand, the preferential darkening of the film emulsion in the described localities disappeared almost completely in preparations with fully developed cataract. Fine lines of blackening of the film corresponding to the lens surface behind the equator were the only residues of the dark shell which characterized the autographs of normal lenses. These results obtained with the autographs were comparable to the analytical data recorded in the laboratory of Dr. Dische on human lenses with cataract, in which the difference in the content of adenosine triphosphate between the cortex and the nucleus of the lens vanished.[9] Experiments with radiophosphorus did not provide evidence, therefore, that the permeability and metabolic activity of lenses with early radiation damage were influenced by roentgen irradiation.

In conclusion, then, none of the four groups of experiments with various radio-indicators suggested a change of permeability or of metabolic processes in irradiated lenses antecedent to the development of lens opacities. Early stages of macroscopically visible radiation injuries of the lens were accompanied with increased uptake of Na^{24} and enhanced permeability for I^{131}. Later stages showed, in addition, a depression of the uptake of radiophosphorus and radiopotassium.

SUMMARY

1. Experiments with radiosodium confirmed the results of earlier studies with other indicators by demonstrating an increased permeability of the blood-aqueous

8. Palm, E.: On the Phosphate Exchange Between the Blood and the Eye: Experiments on the Entrance of Radioactive Phosphate into the Aqueous Humor, the Anterior Uvea and the Lens, Acta ophth., 1948, supp. 32, pp. 1-120.

9. Dische, Z.: Personal communication to the authors.

10. Hevesy, G.: Effect of X-Rays on the Rate of Turnover of Phosphatides, Nature, London **158**:268, 1946.

11. Scott, K. G.: A Comparison of Two Levels of Roentgen and Neutron Irradiation of Normal and Lymphomatous Mice, Using Radiophosphorus as an Indicator of Cellular Activity, Radiology **46**:173-175, 1946.

barrier up to six weeks after irradiation of the rabbit's eye with 2,000 r, but this phenomenon did not appear to play an integral part in the pathogenesis of radiation cataract.

2. The uptake of radiosodium by lenses with radiation cataract was greater than the uptake by control lenses. It increased with the progress of the cataractous process, probably in connection with the development of an extracellular phase in the damaged lens.

3. Radiopotassium penetrated rapidly into the normal lens. Four hours after intraperitoneal introduction of the labeled element the ratio of specific activities of the anterior cortical layers and of the aqueous humor was greater than 1.

4. The accumulation of radioiodine in irradiated lenses with initial cataractous changes exceeded the accumulation in normal lenses and suggested an increased permeability of the damaged lens for this indicator.

5. Radiophosphorus accumulated in normal lenses principally at the equator and in the surface layers of the cortex. The autographic pattern expressed the higher metabolic rate of these parts of the lens. The difference between the superficial cortical and the deeper portions vanished in lenses with total radiation cataract.

6. Experiments with radioactive indicators did not lend support to the theory that enhanced permeability of the lens or depressed processes of phosphorylation lead to the development of lenticular opacities produced by roentgen rays but suggested, rather, that these changes accompany, and possibly influence, the progress of the opacification.

ENUCLEATION AND COSMETIC REHABILITATION

Criteria for Evaluation of Current Procedures

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INTEREST in improvement of the cosmetic appearance of the patient after loss of an eye has resulted in the trial of a wide variety of reconstructive maneuvers. A clarification of the objectives of the operation may aid in the evaluation of current procedures.

> The conjunctiva should first be divided with a thin, sharp pointed knife and next all the muscles inserted into the globe; this being done, the point of the knife would be carried cautiously backwards to the posterior part of the orbit, and the optic nerve divided; the nerve will be more easily divided by directing the knife backwards into the orbit on the nasal side of the globe, as the optic nerve is situated nearer on this side.

This quotation is from Cleoburey's [1] original description of an enucleation, which was published in 1826. The operation differed little in the hands of many surgeons during the next hundred years. Members of the medical profession regarded the removal of an eye as an amputation. In spite of the attempts of Mules,[2] Frost,[3] Lang [4] and their successors to improve the cosmetic appearance, the majority of ophthalmologists felt little responsibility beyond the removal of an offending organ. The restoration of the socket for cosmetic prosthesis was of secondary importance. This attitude was indeed justified because of the disappointing movement of a prosthesis even after the perfect reconstruction of the socket which resulted from a successful Mules or Burch [5] operation.

This failure of the socket to transmit much movement to the prosthesis arose from two factors: the limitations imposed on the shape of the prosthesis by the nature of glass, and the fact that the artificial eye makers fitted the front surface of the prosthesis but never saw or knew the nature of the contact between the prosthesis and the mobile stump.

The war precipitated the utilization of methyl methacrylate for prostheses. It became apparent immediately that this plastic material suffered certain important defects, especially allergenic potentialities and a relative softness in comparison with glass. However, the material could be formed into any desired shape and could be reworked. It was recognized by several ophthalmologists that with this

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1. Cleoburey, W.: A Review of Different Operations Performed on the Eyes, London, T. & G. Underwood, 1826.
2. Mules, P. H.: Tr. Ophth. Soc. U. Kingdom **5**:200-206, 1885.
3. Frost, cited by Lang, W.: Tr. Ophth. Soc. U. Kingdom **7**:286-291, 1887.
4. Lang, W.: Tr. Ophth. Soc. U. Kingdom **7**:286-291, 1887.
5. Burch, F. E.: Tr. Am. Ophth. Soc. **37**:272-282, 1939; Am. J. Ophth. **23**:47-51, 1940; Tr. Am. Acad. Ophth. **49**:237-239, 1945.

material it was possible to fashion both the socket and the prosthesis in any manner which would provide the maximum cosmetic correction of the deformity created by removal of an eye.

This experimentation has now proceeded for eight years. So many difficulties have been encountered and modifications of technic devised that an appraisal of progress in this field seems to be timely. An understanding of the defects created by enucleation should facilitate evaluation of the numerous operations which have been offered to correct the deformities. This paper is an attempt to establish criteria by which the various procedures may be judged.

DISPLACEMENT OF ORBITAL CONTENTS CONSEQUENT ON REMOVAL OF THE GLOBE

The two principal flaws in the results achieved by simple enucleations are (*a*) the sulcus in the upper lid and (*b*) the limitation of external rotation of the prosthesis. There have been many attempts to minimize these flaws, but there has been no sound explanation of what happens in the socket to produce these nearly constant faults. The operating table does not lend itself well to anatomic study, and autopsy material lacks the dynamic factors which are responsible for the changes. Therefore an attempt was made to construct a working model of the orbit, using a variety of plastics to simulate the ligaments and fascial planes, korogel® for the delicate elastic Tenon capsule and rubber bands for the extraocular muscles. This crude device adequately demonstrated the forces under consideration, and the movements that occur in the contents of the orbit after removal of an eye became apparent. The anatomic details are familiar to all ophthalmologists.

When the four rectus muscles are divided, one would expect them to retract and so pull the face of the orbital contents backward. Actually, the space evacuated by removal of the eye is partially filled by a forward movement of 10 to 12 mm. of the posterior portion of Tenon's capsule. This surface, which was roughly the inside of a hemisphere, becomes flattened and is also tipped laterally and upward. The explanation of this behavior is evident on a more detailed examination of the physiological factors involved (figs. 1 and 2).

The axis of the muscle cone is nearly horizontal, but it diverges laterally 23 degrees from the sagittal plane. The muscles pass through Tenon's capsule near the equator of the globe, and the sheaths of the muscles are continuous with the capsule anterior to this line. The direction of pull of the individual muscles is largely determined by the area of contact with the relatively incompressible globe. When this is removed, the rectus muscles naturally tend to straighten and retract. This contraction of the released muscles transforms the spherical cavity left by the globe into a truncated cone and tips the axis of the cone 23 degrees laterally. Since each of the rectus muscles diverges from the axis and its insertion into the capsule is anterior, the contraction also causes an eversion of the lips of the capsule. This distortion is exaggerated in the case of the external rectus. Since its direction of pull is 40 degrees from the anteroposterior plane, the retraction and eversion of the lateral border of Tenon's capsule are limited only by the lateral wall of the orbit and the lateral check ligament. It is evident that if this muscle is allowed to heal in this position, contraction of the muscle, by directing the gaze laterally, will only result in dimpling of the conjunctiva behind the outer canthus. The lateral check ligament, running backward from its origin to the position of the external rectus tendon, can be easily seen in the socket of a simple enucleation. The rectus

muscles, therefore, narrow the equator of the cavity, square and evert the margins of the capsule and rotate the cavity laterally 23 degrees.

The action of the oblique muscles is even more important. These muscles are adherent to Tenon's capsule behind the equator, the inferior oblique almost at the posterior pole. It is obvious that contraction of the oblique muscles will pull the posterior portion of the capsule nasally, thus increasing the action of the external rectus in the lateral rotation of the cavity. They also pull the posterior pole of the space forward, tending to obliterate the space. This forward movement in an orbit with normally semifluid contents amounts to 10 to 12 mm. and turns the surface of Tenon's capsule from a hemisphere into a flat surface. This forward movement is accomplished by the displacement of semifluid fat from the superior orbital compartment into the center of the orbit. The loss of fat in the

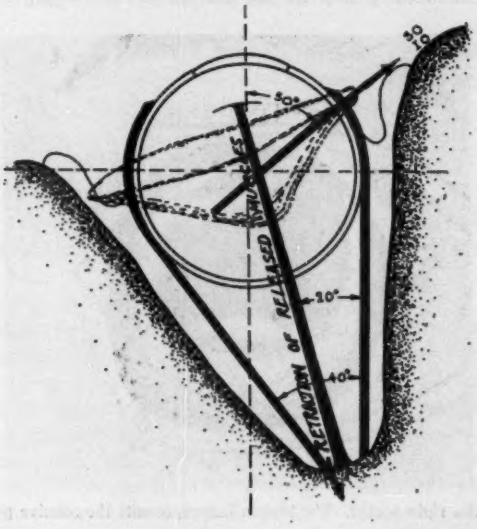

Fig. 1.—Horizontal section of the orbit. The dotted truncated cone shows the relative shape and position of Tenon's capsule after enucleation.

superior compartment results in the deep sulcus in the upper lid, so characteristic of enucleations.

This movement is facilitated by a further action of the superior oblique muscle, contraction of which pulls the capsule upward, as well as mesially and forward. This action should be neutralized by the downward pull of the inferior oblique except for two anatomic factors: (*a*) The downward pull of the inferior oblique is checked by the ligament of Lockwood, which extends from the orbital tubercle beneath the inferior rectus to the mesial wall of the orbit and forms a hammock for the globe; (*b*) the inferior oblique runs beneath the inferior rectus to curl around the globe, and has the longest course in contact with the globe of the extraocular muscles. After removal of the eye, the contraction and straightening of this muscle raises the inferior rectus and, through the fibrinous extensions from the sheath of the inferior rectus to the inferior fornix, tends to obliterate the inferior fornix.

The effect of the superior oblique muscle is well recognized. As fat is forced out of the superior compartment, the tendon of the muscle becomes conspicuous in the upper inner quadrant of the orbit, and nearly all artificial eyes are notched to fit around this cord, which anchors the orbital contents up and forward. The ballooning of the inferior fornix has usually been attributed to orbital hemorrhage or some other factor, but the production of this deformity by release of the inferior oblique muscle is conspicuous in our models.

As soon as the globe is delivered at operation, we produce a cone rotated about 23 degrees temporally, which is displaced temporally and upward and forward. The opening is distorted into a fusiform space running diagonally from the inferior nasal angle to the superior temporal angle by the oblique muscles. The extent of these movements of the socket depends on the influence of inflammatory products and the extent of bleeding into the various tissues, subsequent organization and

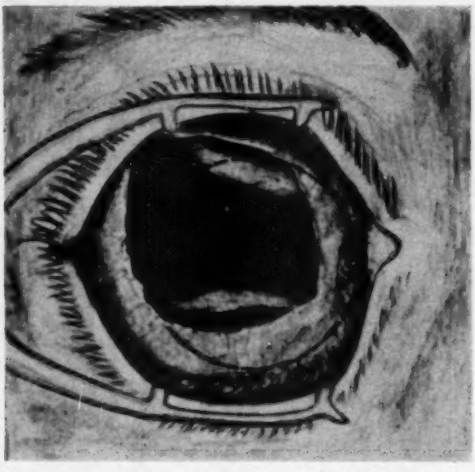

Fig. 2.—Face of a right socket. The broken line represents the relative position of the globe before enucleation.

such modifications as are accomplished by implants or other maneuvers after the removal of the globe. It is logical to assume that this displacement of orbital contents occurs in any reconstruction to the extent that the implant and prosthesis differ in shape, size and center of gravity from the globe which they replace.

THE IDEAL SOCKET

With an understanding of the contractures and displacements that occur in the orbit after removal of an eye, one is in position to postulate the criteria for a successful reconstruction of the orbit.

1. The volume of the globe is 6 to 7 cc., about one fifth of the orbital contents. The usual artificial eye occupies scarcely more than 2 cc. The total loss of 4 to 5 cc. produces a sunken socket and a limitation of motion of the partially retracted rectus muscles. Accurate replacement of volume removed must be a primary object of orbital reconstruction.

Criterion 1: The volume of the implant and prosthesis should approach 6.5 cc.

2. If a plane is passed from the superior bony margin of the orbit to the inferior orbital margin, it passes through the globe in a plane approximately 1 mm. behind the pupil, or about 8 mm. anterior to the center of the globe. The tangential application of the force of the rectus muscles is responsible for the rotation of the globe. If the center of gravity of the eye were gradually moved forward, an increasing fraction of the force of the rectus muscles would be expended in retraction of the globe, and diminished rotation would result.

Restoration of the position of the upper lid necessitates replacement of the anterior surface of the prosthesis in the plane formerly occupied by the anterior surface of the cornea. Restoration of the function of the rectus muscles requires that the center of gravity of the implant and prosthesis lie behind the point of attachment of the muscles to the implant and close to 8 mm. behind the plane of the orbital rim.

Criterion 2: The center of gravity of the implant and prosthesis must be within the muscle cone and should lie 8 mm. behind the anterior orbital plane.

To these new criteria should be added those long considered.

Criterion 3: The four rectus muscles and, if possible, the two oblique muscles should be attached to a mobile stump or implant so that they do not atrophy.

Criterion 4: No avenue for infection should be left.

Criterion 5: The orbit should not be obscured by dense scar tissue or an implant in cases in which local recurrence of a neoplasm is to be anticipated.

EVALUATION OF PRESENT OPERATIONS

Simple Enucleation.—The socket of a simple enucleation undistorted by organization or exudate or hemorrhage is well known. Whether the conjunctiva is sutured or not, the socket epithelizes, forming a slightly convex surface tipped temporally about 25 degrees from the vertical plane, extending well in behind the lacrimal gland but narrowed sharply in the upper inner angle by the superior oblique muscle and often showing a ballooning upward of the inferior fornix by the contraction of the inferior oblique muscle. Because of the displaced position of the muscle cone, contraction of the external rectus results largely in a retraction of the temporal border of the face of the socket. Even so, this plane is often fairly mobile and will impart some motion to a prosthesis which presents a deeper curvature on the posterior surface than the surface of the socket, so that it rests on the edge and tends to force the muscle cone into the prosthesis. This surface of the prosthesis should also be decentered upward and outward from the anterior surface of the prosthesis. The most mobile prosthesis for simple enucleation is a combination of a spherical anterior surface of about 25 mm. diameter with a cup on the posterior surface the axis of which is tipped temporally 22 degrees and 10 to 20 degrees upward. The axis of this cup should lie 5 to 10 mm. up and out from the position of the pupil. These requirements result in a prosthesis which is virtually a shell on the inferior nasal edge, becoming thicker superiorly and temporally.

If the surgeon ties the tendons of the four rectus muscles together, he obtains a small organized mass in the superior temporal quadrant of the socket, which does tend to keep the muscles from atrophying and which improves the movement of the prosthesis slightly if the same suggestions in fitting are followed.

Spherical Implants.—A 10 mm. ball, once recommended by oculists, has a volume of 0.4 cc. The replacement of tissues is negligible, and this small lump in the superior temporal quadrant of the socket, even though it keeps the muscles from becoming atrophic, has little effect either on the movement of the prosthesis or on the sulcus in the upper lid. About all it accomplishes is to mark the position of the muscle cone. The position in the superior temporal quadrant of the socket does not necessarily mean that the implant has migrated out of the muscle cone. The small implant does little to overcome the displacement of tissues consequent on the removal of the globe, and such a socket is often fitted with the same reform eye as that which would be used if the implant had not been inserted.

A 14 mm. ball has a volume of 1.1 cc., and an 18 mm. ball, a volume of 2.3 cc. There seems to be little reason for using an implant smaller than 18 mm. Even a 20 mm. sphere replaces 3.2 cc., only half the volume removed, and it will not be centered in the socket. The posterior surface of a prosthesis should be correspondingly displaced upward and outward.

Evisceration.—Simple evisceration results in shrinking and flattening of the remaining sclera, so that the socket eventually resembles that of a small implant except that the face of the socket is flat and the mobile stump is easier to disregard in fitting with a glass eye than is the rounded protuberance of an implant.

Mules's operation involves some loss of volume because of the small size of the implant it is practicable to place within the sclera after the cornea has been removed. This leads to a corresponding displacement of the implant upward and outward.

With a 16 or an 18 mm. implant in the sclera, a very fine mobile, thin shell prosthesis can be fitted. Whenever it is practicable, this may well be considered the operation of choice unless the surgeon has access to the facilities of a prosthetic laboratory, so that he can utilize the newer operations.

Burch's [5] evisceration, in which the cornea is retained, fulfils all the present criteria, as it allows the insertion of an 18 to 20 mm. ball within the fibrous tunic and the stump remains well centered in the orbit. Unfortunately, it is not often a feasible operation for the group of young people for whom a cosmetic result is important, since it cannot be used with a shrunken globe or a severely damaged globe or in the case of suspected tumor, and late extrusion of the implant, due to erosion of the cornea,[6] has been reported.

When any large implant is to be placed within the sclera, a large opening in the posterior segment should be fashioned first, as advocated by Huizinga.[7] Some late contracture of the sclera does occur, although this is difficult to explain. A posterior opening allows the sclera to contract anteriorly. If the sclera is closed posteriorly, there may be reopening of the anterior scar with late contracture. I have not lost a scleral implant since I began to utilize the posterior opening, about 10 years ago.

Semiburied Implants.—Guyton [8] has reviewed the voluminous literature on recent developments in the use of semiburied implants. Students of the subject

6. Hughes, W. L.: Am. J. Ophth. **31**:303-310, 1948. Dimitry, T. J.: South. M. J. **10**:594-600, 1917.

7. Huizinga, J. G.: Eviscero-Neurotomy: A New Operation, J. A. M. A. **34**:394-395 (Feb. 17) 1900.

8. Guyton, J. S.: Am. J. Ophth. **32**:1517-1531, 1949; **32**:1725-1734, 1949; **33**:283-290, 1950.

are referred to this comprehensive treatise for descriptions of the various procedures which have been attempted. Comment here will be limited to the application of the proposed criteria to these operations and the effect of experience with semiburied implants on the criteria.

Original fears of infection and extrusion of semiburied foreign bodies appear now to be only partially justified. With certain implants, or "exoplants," as Guyton asserts, this may be due to epithelization of the surfaces of contact. However, with other types, such as those of Whitney and Olson,[9] Stone [10] and Cutler,[11] epithelization can scarcely occur. Successful retention of such implants seems to depend on smooth, impervious adhesions between the conjunctiva and the tantalum ring surrounding the neck. These implants are not extruded unless tantalum is exposed. Late exposure may be due to pressure from improper correlation of the prosthesis and the implant or to fragmentation of the tantalum mesh or sutures. Once tantalum is exposed, profuse, intractable discharge and infection appear. The conjunctiva slowly retracts, and a muscle tendon, usually the superior rectus, becomes exposed and necrotic. Even under these conditions the adhesions between the tendon and the tantalum mesh do not separate. Slough of the tendon behind these adhesions occurs.

This sequence is scarcely a typical foreign body reaction. The explanation appears to include several factors: the combination of materials used, the utilization of tissues, the size and shape of the implant and the coaptation of prosthesis and socket.

Materials: The principal materials used in semiburied implants have been methyl methacrylate, tantalum, vitallium® and gold. Allergic reaction of the acrylic and chemical irritation attributed to incomplete polymerization have been blamed for late extrusions. Certainly, the implant should be inert and impervious to water. Even under the most advanced methods of manufacture there must be some fluid exchange between a methyl methacrylate implant and the surrounding tissues. Other plastics are certainly more inert. The best appears to be KEL-F, with which Jardon and Ruedemann [12] are presently experimenting.

However important this factor, implants of acrylic alone or of acrylic and gold do not cause as troublesome a discharge as do those of tantalum and acrylic. Cutler ring implants were often lost; but, as will be explained later, this was due to other factors, and discharge was not a problem, though gold and acrylic were both exposed for a long time. Guyton does not list discharge as a complication of his implant. I have experimented with various gold and gold and acrylic semiburied implants. In my hands the most successful semiburied implants was the "may basket" model (fig. 3). The muscles were tied by a purse string suture about the gold neck. Tenon's capsule was sewed by a double mattress suture over the face of the implant, and the gold handle protruded through the conjunctiva, which was closed horizontally. A flexible plastic fin attached to the back of the prosthesis absorbed strain on the muscle attachments at extremes of rotation. Five of these

9. Whitney, E. L., and Olson, J. A., cited by Guyton.[8]

10. Stone, W., Jr.: Preliminary Presentation at the Scientific Exhibit, Ninety-Sixth Annual Session, American Medical Association, Chicago, June 21-25, 1948.

11. Cutler, N. L.: Influence of Local Antiseptics on Regeneration of Corneal Epithelium of Rabbits, Arch. Ophth. **35**:71-83 (Feb.) 1946.

12. Jardon, F., and Ruedemann, A. D.: Personal communication to the author.

implants were inserted in 1948, and the cosmetic results are comparable to those of other direct contact implants. None of the patients remove their prostheses, and none have more than normal mucous secretion from the socket. A 0.25 per cent zinc sulfate solution is used once or twice daily. These encouraging experiments were discontinued, pending the development of a gold or plastic mesh to cover the face of the implant, in favor of experiments with a buried implant. The experience is detailed to illustrate the difference between exposures of gold and of tantalum in the socket. Guyton and Stone have recognized that tantalum mesh and tantalum sutures promote a firm fibrous union, which is not produced by gold or vitallium.* If the tantalum mesh has been extended behind the actual point of desired attachment, late limitation of motion of the implant results. The use of tantalum is also complicated by the tendency to fragmentation. This is a serious hazard when

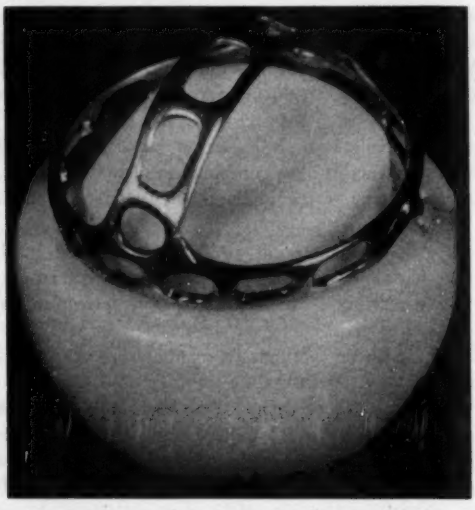

Fig. 3.—May basket implant. Only the gold handle is exposed in the socket. Integration is obtained through a flexible plastic fin on the back of the prosthesis.

muscles are sutured to tantalum mesh. Stone and Ruedemann have both discontinued this practice in favor of pins and foot plates.

Tissue Utilization: If the conjunctiva is utilized, it must be placed firmly in position by tension and pressure, else it loses its character and becomes thick, tough, inelastic and possibly infected.

Tenon's capsule is very elastic. It offers an ideal receptacle for an implant, since it will adapt itself to any reasonable size or shape and retain its character in contact with a polished surface. Fralick [13] and Ellis [14] stated that an 18 mm. sphere is the largest implant over which Tenon's capsule may be closed. There is basis for fear of extrusion of large implants when the muscles are not attached to the implant,

13. Fralick, F. B.; McCotter, R. E., and Henderson, J. W.: Tr. Am. Acad. Ophth. **52**: 1949.
14. Ellis, O. H.: Am. J. Ophth. **32**:990-992, 1949.

as is practiced with a glass sphere. However, if the muscles are utilized there is no hazard of posterior extrusion. The position of the implant depends on other factors, and Tenon's capsule is sufficiently elastic that it need not determine the size of the implant.

There remains the utilization of new-formed tissue. If Tenon's capsule is not closed, a fibrocytic pseudomembrane replaces the endothelium. This membrane is somewhat resistant to infection.

According to Guyton, epithelization of the entire exposed surface of tissues may be promoted if there is no direct attachment of the tissues to the "exoplant."

Size and Shape of the Implant: It is doubtful whether any other operation can approach the immediate cosmetic result obtained with Ruedemann's original operation, in which there were nearly anatomic replacement of volume removed and physiologic action of the muscles. Anatomic replacement in the case of an integrated implant means that the prosthesis and implant should approach a sphere 24 mm. in diameter. Each digression from this size and shape means that a countermeasure must be introduced to counteract the deformity produced.

The volume of an artificial eye is 1.5 to 2.5 cc. If one is replacing a 6.5 cc. globe, the ideal volume of the implant is about 4.0 cc. The volumes of semiburied implants in current use are as follows:

	Volume, Cc.
Stone: Lateral edge	1.6
Stone: With pins	2.2
Whitney and Olson	2.6
Guyton	1.8
Cutler	1.9
Ruedemann (new implant)	3.0

Whatever the volume of implant plus prosthesis, only the implant lies within the muscle cone, and the positioning of this implant in the orbit depends on the volume of the implant and where the muscles are sutured. The volume of a 17 mm. sphere is 1.9 cc., and attachment of a pin to the anterior surface will scarcely prevent retraction to the temporal wall of the orbit and limitation of external rotation.

The circumference of the average eye is about 76 mm. The anterior distance on an arc between the insertion of opponent rectus muscles is 24 to 25 mm. It follows that the posterior circumferential distance between the point of insertion of the external rectus and the point of insertion of the internal rectus (and likewise those of the vertical rectus muscles) is about 52 mm. The circumference of a 17 mm. sphere is 53.4 mm., which means that with the size of implants used by Cutler,[11] Stone[10] and Whitney and Olson[9] the muscle tendons should be sewed together over the implant if the posterior pole of the implant is to lie in the position in the orbit formerly occupied by the posterior pole of the eye. A 4 or 5 mm. recession of all of the rectus muscles cannot be disregarded. Guyton's technic places the posterior pole of his "exoplant" in the proper theoretical position.

As to the shape of the implant, there seems to be no reason for changing the posterior segment from that of 25 mm. sphere. How far forward can this shape be carried? The muscles are attached to the eyeball approximately 18 mm. anterior to the posterior pole. This plane forms a circle 20 mm. in diameter. The anterior pole of the prosthesis must lie 7 mm. in front of this plane. Obviously, a 25 mm.

sphere sliced off in this plane would not leave sufficient space for the prosthesis. Reduction in volume would seem to be accomplished best by reduction in the diameter of the anterior portion, rather than by over-all reduction in the size of the sphere, in order to maintain the tangential action of the rectus muscles and to maintain a center of gravity behind the plane of attachment of the muscles. A possible theoretical solution is a hemispherical implant 24 mm. in diameter with the muscles attached to a circle 12 mm. in diameter. The prosthesis would then take the form of the other half of the hemisphere. There are no theoretical limits on the design of the anterior segment except that the prosthesis and the face of the socket should fit.

Reduction of the size of the posterior segment allows forward movement of the orbital contents and moves the center of gravity forward. Guyton's "exoplant" is an extreme example of this type. The face of the prosthesis moves about twice as far as the muscle insertion. The posterior pole of the exoplant acts as a fulcrum, as Guyton has pointed out, and the movement of the artificial eye must be a displacement rather than a rotation.

Coaptation of Implant and Prosthesis: In all integrated implants the force of the muscles is transmitted directly to the prosthesis. This is a great advantage, but it also means that shock and strain of movement of the prosthesis against any obstruction falls on the adhesions between the tendons and the implant. Guyton has adequately compensated for this by the tissue to tissue attachment of the muscles, but this strain and the pinching of conjunctiva due to uneven relations between prosthesis and implant are probably responsible for the late exposure of tantalum in implants of the Cutler and Stone types. In buried and in semiburied implants it is not sufficient that the prosthesis be integrated by a pin. A good design takes into consideration the prosthesis as well as the implant. A smooth fit in all fields of movement is necessary, since areas of pressure lead to erosion and intervening areas show fibroblastic proliferation. Obstruction of movement by intervening tissues endangers the attachments of the tendons to the implant. A prime requirement of a satisfactory reconstruction, often disregarded in the past few years, is a socket which can be fitted. There can be no objection to unanatomic reconstructions provided that proper compensation is made for defects introduced. Cosmetic rehabilitation is not a problem to be solved by a single procedure. An armamentarium of several types of implants is needed. McDowell [15] has devised a gold bar "exoplant" which is applicable to the scarred and contracted socket.

Buried Implants.—Hoffman [16] modified the Cutler ring implant by reducing the acrylic sphere and covering the front of the implant with tantalum mesh. This buried implant provided a closed socket. However, the small replacement of volume allowed retraction of the muscles. Prostheses fitted in our laboratory have been disappointing in range of motility.

Allen and Allen [17] have experimented for several years with buried implants and have developed a tunneled hemispherical implant. The muscles are quite properly

15. McDowell, F.: Am. J. Ophth. **33**:953-958, 1950.

16. Hoffman, L. G.: Description and Surgical Technique Using a Hoffman Inclusion Implant, Chicago, Mager & Gougelman, Inc., 1948.

17. Allen, J. H., and Allen, L.: A Buried Muscle Cone Implant: I. The Development of a Tunneled Hemispherical Type, Arch. Ophth. **43**:879-890 (May) 1950.

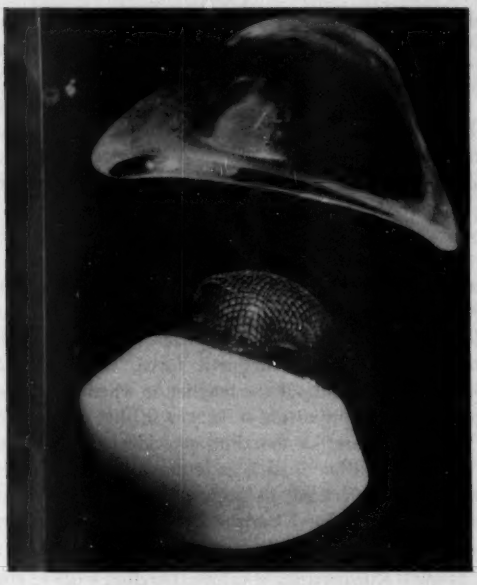

Fig. 4.—Lateral view of buried implant, with transparent perforated temporary prosthesis to be inserted at completion of operation.

Fig. 5.—Coaptation of implant and prosthesis. Prosthesis and implant approximate a sphere 25 mm. in diameter.

sutured together, as with Guyton's implant. The capsule and conjunctiva are closed over the anterior surface. The prosthesis is nearly hemispherical. On a theoretical basis there is little choice between this procedure and my buried implant.

Troutman [18] made an important contribution in his experiments with the magnetic implant. He has utilized a 17 mm. spheroid flattened anteriorly. A magnet is embedded beneath the face of the implant. A 3 mm. ring of tantalum mesh surrounds the anterior rim. A purse string silk suture is threaded through the muscle tendons and tied about the tantalum ring. Tenon's capsule and conjunctiva are closed separately over the implant. The prosthesis is equipped with a magnet, so that movement is imparted to the prosthesis by magnetic force. Our experience with this implant has been both stimulating and disappointing. The small size of the implant allowed retraction, as with Hoffman's implant. In addition, the bulk and weight of the prosthesis necessary for inclusion of the magnet have nullified the advantage of the utilization of magnetic force. We have obtained as good motion with prostheses fitted without the magnet as when it is included. However, Troutman has provided two important advances in this experimentation. He has discovered that a narrow band of tantalum provides a better attachment for the muscle tendons than a broad band, such as is used by others, and that suturing to the tantalum mesh, with the hazard of fragmentation of the tantalum, is unnecessary. Both of these principles have been utilized by us in our current procedure.

I have experimented with a variety of reconstructions of the orbit during the past eight years, as have Ruedemann, Whitney, Guyton, Cutler, Hughes and others. Beautiful cosmetic results are certainly being obtained with certain semiburied implants when used in conjunction with meticulous surgical technique and aftercare. It is hoped that such results will stimulate further development of the possibilities of enhanced movement of the prosthesis by direct contact with an implant. However, the complications still prevalent in the hands of the average surgeon have led many to seek an operation which gives better results than a glass ball implant without the difficulties of a semiburied implant.

The present procedure is offered to meet these needs. The implant is 23 mm. in diameter and measures 21 mm. anteroposteriorly. A neck 11.5 mm. in diameter is provided for the positioning of the rectus tendons. This, and the face of the implant, are covered with a double tantalum mesh. The implant has a volume of 4.0 cc. The center of gravity of prosthesis and implant lies behind the insertion of the muscles. The size and shape have been evolved from theoretical considerations so that the implant and prosthesis may approximate the globe in size, position and center of gravity. There is satisfactory coaptation between the face of the socket and the prosthesis (figs. 4 and 5).

PROCEDURE

The conjunctiva is divided at the limbus and dissected well back. Tenon's capsule is divided at one border of the insertion of the internal rectus and a muscle clamp slipped over the tendon, which is then excised from the globe. Tenon's capsule is divided with scissors as far anterior as possible if it is not too thin. Each rectus muscle in turn is clamped and divided, the four clamps being kept on the tendons and laid backward while the globe is delivered. The use of a tonsil snare to divide the nerve reduces bleeding after removal of the globe.

18. Troutman, R. C.: Description and Surgical Technique Using an Integrated Magnetic Implant, Chicago, Mager & Gougelman, Inc., 1948.

In any case, bleeding must be stopped if necessary by tying bleeders before the implant is inserted. A no. 1 white silk suture is threaded through each rectus tendon just behind the blades of the clamp. A single whip stitch through one edge of the tendon, a bite in the center and a single whip stitch through the other border of the tendon, leaving about 2 mm. of suture material between tendons, is the current practice. The implant is held with a curved hemostat by an assistant and inserted into Tenon's space while the purse string suture is tied about the neck. The edge of Tenon's capsule is closed diagonally over the implant with double mattress 4-0 surgical gut sutures. The conjunctiva is closed with a single running silk suture. A temporary prosthesis is inserted and pressure bandage applied. Pressure is maintained for 10 days. If the dressing is changed in the interim, the socket may be examined and irrigated, if necessary, through the holes in the temporary prosthesis.

The permanent prosthesis can be fitted in two weeks. The outer surface should be practically hemispherical and 25 mm. in diameter, and needs no notch for the superior oblique muscle. The implant is in nearly the position of the eye, and in the center of the posterior surface of the prosthesis a shallow cuplike depression should be ground out to receive the ball-like protuberance from the implant.

Twenty-three implants of this type have been inserted by several surgeons during the past two years. Two early implants were lost. The loss in one case was attributed to slipping of the muscle to one side over the equator of the implant. The shape of the implant was modified slightly, and this has not occurred again. The other extrusion was caused by a hematoma which formed behind the implant. More care in hemostasis and firm pressure seem to have eliminated the danger of this complication.

The replacement of tissues with this operation results in a good cosmetic reconstruction. No sulcus appears in the upper lid. Movements are nearly normal in all directions except temporally, where there is some lag of the prosthesis in extreme rotation. Within the range of fixation with glasses the prosthesis follows very well. No late sequelae, such as troublesome discharge, have been encountered.

SUMMARY

The following criteria for evaluation of reconstruction following enucleation are suggested:

Criterion 1: The volume of the implant and prosthesis should approach 6.5 cc.

Criterion 2: The center of gravity of the implant and prosthesis must be within the muscle cone and approach a position 8 mm. behind the anterior orbital plane.

Criterion 3: The four rectus muscles and, if possible, the two oblique muscles should be attached to a mobile stump or implant so that they do not atrophy.

Criterion 4: No avenue for infection should be left.

Criterion 5: The orbit should not be obscured by dense scar tissue or an implant in cases in which local recurrence of a neoplasm is to be anticipated.

A buried implant which satisfies these criteria is offered. Results with the operation show a good cosmetic appearance, with the prosthesis following the fellow eye within the range of fixation allowed by glasses. No troublesome sequelae have been encountered.

The United States Navy and Ohio State University provided experimental prosthetic laboratories and technical assistance since 1943, and several volunteer and professional technicians have contributed to this study. James Guliano, at the Portsmouth Naval Hospital, and James Bitonte, at Ohio State University, have given particularly valuable collaboration.

AMAUROSIS FUGAX ET AMAUROSIS FULMINANS

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AMAUROSIS FUGAX

IF ONE were to look up the word "blackout" in the Oxford Dictionary, one would not find it there. This word, which so aptly describes "amaurosis fugax," and with which we have become familiar during the two wars, is a newcomer. "Blacking out," which later became "blackout," was a term introduced to describe a form of temporary blindness which became an increasingly frequent occurrence in flying. The sudden change in direction of flight at high speed when the pilot is pulling out of dives or taking sharp turns sets up centrifugal forces of such magnitude as to produce far reaching physiological effects. During World War II these effects were subjects of intensive investigation, which was carried out with the aid of enormous centrifuges provided with cockpits free to move outward when the centrifuge is in motion.[1] Since the speed of rotation and the radius are known, the magnitude of the centrifugal force can readily be calculated. The effect of the centrifugal force is to increase the weight of the body, and therefore the centrifugal force is expressed in multiples of earth's gravitational force. For example, the pilot would experience a force of 4 g while making a turn with a radius of 1,500 feet (455 M.) at a speed of 300 miles (482 kilometers) per hour. The pilot's weight would become quadrupled, and it would be barely possible for the legs and arms to be lifted. Manifold effects result from this increase in weight. A pressure of 25 mm. of mercury is required to overcome the normal force of gravity in order to raise blood from heart to head level, a vertical distance of about 0.3 M. At a force of 4 g the pressure required will be about 100 mm. of mercury. If the subject's systolic arterial pressure at heart level is 120mm. of mercury, the pressure of blood at head level during exposure to a force of 4 g will be 120 — 100 mm. of mercury. Since the circulation of the retina is opposed by a normal intraocular pressure of approximately 18 mm., it is clear that retinal blood flow will cease under these conditions. After exposure to 3 g for three seconds, loss of peripheral vision occurs. At 4 g, after a similar interval, there is complete loss of vision without disturbance of hearing, smell or consciousness; i. e., true blackout occurs. Diminution of hearing and, later, loss of consciousness occur during exposure to a force 0.5 g greater than that required to produce blackout.

This paper was given as a lecture before the Institute of Ophthalmology, University of London.

1. Stewart, W. K.: Some Observations on Effect of Centrifugal Force in Man, J. Neurol., Neurosurg. & Psychiat. **8**:24, 1945. Lambert, E. H.: Site of Origin of Blackout in Aviators, Proc. Am. Federation Clin. Research **2**:64, 1945; Clin. Investigation **25**:912, 1946.

Blood pressure measurements at head level were carried out by puncturing the radial artery, with the subject's wrist supported at head level, and making a continuous record of the pressure. It was found that complete loss of vision occurred when the systolic pressure was just under 20 mm. of mercury. Unconsciousness occurred when systolic pressure had fallen to zero. The retinal origin of blackouts was also confirmed by experiments in which 30 to 40 mm. of mercury suction was applied to the eyeballs by means of a special mask. With such a mask consciousness was lost without a preliminary blackout when the subject was exposed to 4.5 g. Similarly, when 20 to 30 mm. of mercury positive pressure was applied to the eyeball, this lowered by 1 g the force necessary to produce a blackout.

The effect of centrifugal force on blood pressure at heart level is remarkably slight. With the wrist supported at the level of the base of the heart, the systolic pressure measured within the radial artery decreased on the average 5 mm. of mercury for each additional g of force. This fall is due to pooling of blood in the veins of the legs. Compensation quickly follows, and blood pressure at heart level may increase by 20 to 70 mm. of mercury above the initial value after a few seconds. The phenomenon of blackout was also studied experimentally by Rossen, Kabat and Anderson.[2] By means of an inflatable cervical pressure cuff, held down to the lower third of the neck, and by raising the pressure to 600 mm. of mercury within one-eighth second, they studied the effect of sudden arrest of cranial circulation. Many subjects experienced narrowing of the field of vision, blurring of vision, with the field of vision becoming gray, and, finally, complete loss of vision. A number of subjects stated that for a short time they were unable to see but could still hear before losing consciousness.

The visual fields under conditions of oxygen deficiency were studied by Evans and McFarland.[3] They found obliteration of peripheral fields with preservation of central visual acuity even at the peak of oxygen deprivation. Air Commodore Livingston also stated in his Moynihan lecture[4]:

> It is exceptional for visual acuity recorded as 6/6 under normal circumstances to fall below 6/9, although the subject may show signs of severe anoxaemia.

This led Livingston to the conclusion that the retinal cones meet anoxemia with greater powers of resistance than the rods. Yet in the area of retina where the density of the cone population is maximal, i. e., in the center of the fovea, no blood vessels are detectable by histological means. Dartnall and Thomson, of the Vision Research Unit of the Institute of Ophthalmology, London, postulated, therefore, that this part of the retina is supplied by a secondary oxygen-carrying system, since it is unlikely that the oxygen needs of this portion of the retina are less than those of the rest of the retina. They suggested[5] that the yellow pigment of the fovea may be concerned with the oxidation mechanism and may provide oxygen to this retinal region.

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2. Rossen, R.; Kabat, H., and Anderson, J. P.: Acute Arrest of Cerebral Circulation in Man, Arch. Neurol. & Psychiat. **50**:510 (Nov.) 1943.

3. Evans, J. N., and McFarland, R. A.: Effects of Oxygen Deprivation on Central Visual Field, Am. J. Ophth. **21**:968, 1938.

4. Livingston, P. C.: Visual Problems of Aerial Warfare: "Night"; Studies in Dark-Adapted Eye, Lancet **2**:33, 1944.

5. Dartnall, H. J. A., and Thomson, L. C.: Retinal Oxygen Supply and Macular Pigmentation, Nature, London **164**:876, 1949.

It can be concluded, therefore, that blackout will occur whenever the cerebral blood pressure falls below the intraocular pressure and that it will be most obvious when the fall of blood pressure takes place gradually. The main causes of such a fall in blood pressure are (1) the common faint, (2) an overactive carotid sinus and (3) the Stokes-Adams syndrome.

Perhaps the extremely rare cases of pulsating darkness associated with aortic insufficiency reported by Bittorf [6] may be due to the same mechanism. The commonest cause of recurrent "blackouts" is migraine. Recent experimental work has given some insight into the mechanism of visual disturbances occurring in migraine.[7] Since they can be abolished by inhaling amyl nitrite in amounts insufficient to produce a drop in blood pressure, it can reasonably be inferred that they are caused by vasoconstriction. Furthermore, the homonymous distribution, and perhaps the association with crude visual hallucination, can be regarded as providing evidence that such vasoconstriction involves the posterior cerebral arteries. Personally, I feel that in most cases of migraine in which obscuration of vision alone is the chief manifestation the visual disorder is the result of vasoconstriction of retinal arteries, rather than of cerebral arteries. This conclusion is supported by the following evidence:

1. The disturbance of vision is frequently uniocular.

2. Occasionally such an episode is not followed by the customary complete recovery, and then ophthalmoscopic examination reveals occlusion of one or more branches of the central retinal artery in the absence of any disease of the retinal vessels.[8] Such thrombosis in my experience is oftenest seen in young women with anemia.

The usual manifestation in these cases of transitory amaurosis is bilateral obscuration of vision recurring in attacks of varying duration and not followed by headache. The condition is usually regarded as hysterical. Occasionally, presumably only branches of the central retinal artery are picked out, and the distribution of defects is then remarkably constant in any given patient. I had a case in which the defect was of inferior altitudinal type when the left eye was affected and central when the right eye was involved. It is, of course, quite possible that the visual disturbance in some patients is the result of the combined involvement of retinal and cerebral arteries. The symptoms of congenital hemangiomatous malformations of the brain known as arteriovenous aneurysms, when occurring in the occipital region, may mimic migraine very closely, as is illustrated by the following case:

Since the age of 21 this woman, who at the time of consultation was 46, had been troubled with frequent attacks of obscuration of vision, when she was not able to see anything clearly. Usually the vision cleared in a matter of minutes. Occasionally these episodes were followed

6. Bittorf, A.: Über pulsatorische Verdunkelung beider Gesichtsfelder, bei Aorteninsuffizienz, Klin. Monatsbl. f. Augenh. **70**:399, 1923.

7. Schumacher, G. A., and Wolff, H. G.: Experimental Studies on Headache: Contrast of Histamine Headache with Headache of Migraine and That Associated with Hypertension; Contrast of Vascular Mechanisms in Preheadache and in Headache Phenomena of Migraine, Arch. Neurol. & Psychiat. **45**:199 (Feb.) 1941.

8. Grönvall, H.: On Changes in Fundus Oculi and Persisting Injuries to Eye in Migraine, Acta ophth. **16**:602, 1938.

by the appearance of "flashes of lightning," which generally heralded a severe headache, most frequently on the right side of the head. On a half-dozen occasions these attacks were followed by loss of consciousness. Three months prior to consulting me, she experienced an intense headache, which she described as "pumping, as though my head was going to burst"; at the same time she saw "flashes of lightning." This attack was associated with a subarachnoid hemorrhage. An arteriogram revealed a typical arteriovenous aneurysm in the right occipital lobe.

Recurrent vasoconstriction of the retinal arteries is, of course, also commonly seen in cases of hypertensive vascular disease with retinal arteriosclerosis. There remains a small group of cases of amaurosis fugax, usually recurring in the same eye, among persons without migrainous traits and without evidence of hypertensive vascular disease. Usually this condition is regarded as retinal angiospasm. Most clinicians will agree that retinal angiospastic manifestations are to be expected as part of a general vasoconstrictor response associated with a rise in blood pressure, e. g., to tobacco or cold. It is also generally accepted that such retinal angiospasm may occur as a result of exposure to exogenous poisons, such as lead or quinine. However, in a number of cases all these factors can be excluded. I believe it is open to criticism, despite the clear ophthalmoscopic evidence of transient cessation of retinal circulation, to assume that in every such case the primary cause is retinal. It is clear that if the pressure in retinal arteries is less than the intraocular pressure collapse of these vessels will take place even when the cause is retrobulbar, i. e., in the ophthalmic artery. It is well known that Raynaud's phenomenon may occur solely as a result of narrowing of an artery, e. g., as a result of disease involving the intimal coat. Normal increase in vascular tone resulting from cold, pain or emotion is under these conditions capable of interfering to a high degree with the blood stream through the affected artery. Localized atheromatous changes in arteries are not uncommon even in persons without any evidence of generalized vascular disease.[9] Carotid arteriograms have revealed unsuspected occlusion of the internal carotid artery. I would postulate an occluding pathological process affecting the internal carotid or ophthalmic artery as the explanation in some of these cases of amaurosis fugax. Mayou mentioned a case of recurrent amaurosis of one eye in which gross evidence of syphilis of the cardiovascular system was found at autopsy. Most cases of this condition in my experience and those described in the literature have shown spontaneous remission, and this might be accounted for by the establishment of an adequate collateral circulation. One may hope that arteriography will in future advance the understanding of this condition. Before leaving the subject of ocular angiospasm, I must mention that some authors have postulated spasms of capillary vessels of the retina or choroid to explain serous central retinopathy.[10] The evidence for this hypothesis is indirect and extremely slender.

A number of other causes of amaurosis fugax remain to be discussed.

Papilledema. Occasionally patients with bilateral papilledema complain of sudden recurrent, painless and bilateral attacks of amaurosis of brief duration. In some patients these attacks occur only when the head is in certain positions. In

9. Abrahams, A.: Arterial Calcification: A Personal Observation, Lancet **1**:395, 1950.

10. Gifford, S. R.: Evaluation of Ocular Angiospasm, Arch. Ophth. **31**:453 (June) 1944. Duggan, W. F.: Choroidosis Centralis Serosa: Diagnosis, Pathologic Physiology and Therapy, ibid. **27**:123 (Jan.) 1942.

view of the simultaneous involvement of vision in both eyes, it can be argued that amaurosis is due to pressure on the optic chiasm, presumably by a ballooned third ventricle.

Closed head injuries. Such injuries may produce transient amaurosis in a number of ways. The optic nerves are relatively fixed structures which anchor the brain and are liable to suffer damage by change of velocity imparted to the head by a blow. This can result in transient unilateral or bilateral blindness.

After head injuries, it is not uncommon for migrainous tendencies to become exaggerated, and this may give rise to the appearance of amaurosis fugax of migrainous type. Cases of this nature have been recorded in the literature, and I myself have seen several instances.

Epilepsy. A phenomenon not uncommon in epilepsy is Todd's paralysis (post-convulsive paralysis), which affects the part most involved during the seizure. It may be regarded as due to exhaustion of the ganglion cells, and normal function is as a rule quickly recovered. The phenomenon is oftenest seen in children. If the occipital cortex is the main site of the epileptic discharge, transient hemianopsia or transient total amaurosis may be encountered.[11]

AMAUROSIS FULMINANS

Bilateral blindness is usually the result of bilateral lesions, e.g., bilateral simultaneous hemorrhage into the vitreous in the case of subarachnoid hemorrhage. The two visual radiations, however, gradually approach each other as they proceed caudally, and, finally, in the calcarine region they are only 5 mm. apart, separated from each other by the falx cerebri. In this situation, as also in the chiasmatic region, a single comparatively small lesion is capable of producing simultaneous bilateral blindness. However, before attaching diagnostic significance to the simultaneous bilateral onset, it is important to exclude preexisting unilateral amaurosis or homonymous hemianopsia which may have passed unnoticed by the patient prior to the onset of blindness, and this is by no means an easy matter.

Bilateral Occipital Lesions.—The posterior cerebral artery, shortly after arising from the basilar artery, arches backward and upward between the crus cerebri and the uncinate convolution. During part of its course, it may come close to the free edge of the tentorium. A supratentorial tumor which produces herniation of the uncus through the incisura tentorium is likely to compress the posterior cerebral artery. A not uncommon postmortem finding is cortical necrosis of both occipital lobes of the type which may have been produced by arterial obstruction, presumably in the manner described.[12] As a rule, this condition is not compatible with life, and the condition is of clinical importance only when it occurs on one side, when homonymous hemianopsia may be observed shortly before death.

However, there are a number of cases of vascular lesions involving both occipital lobes. Two such cases merit notice.

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11. Harris, W.: Hemianopia, with Especial Reference to Its Transient Varieties, Brain **20**:308, 1897.

12. Moore, M. T., and Stern, K.: Vascular Lesions in the Brain Stem and Occipital Lobe Occurring in Association with Brain Tumours, Brain **61**:70, 1938.

HEMPHILL'S[13] CASE.—A man aged 65, while eating dinner, complained of a sudden intense headache, stood up and said he was blind. He did not lose consciousness, but for a few hours he was slightly confused and did not seem to know exactly where he was. The pupillary reflexes were normal; the blood pressure was 220/100; the cerebrospinal fluid showed nothing abnormal. One week later he was able to distinguish light from darkness in the lower external quadrant of the left visual field. When he died, a year later, it was found that the basilar artery at the origin of the posterior cerebral arteries was firmly occluded by an old organized clot, which extended along each posterior cerebral artery. Both occipital lobes were necrosed.

Thrombosis of the basilar artery rarely gives rise to visual disturbances because the blood supply to the occipital lobes is maintained through the posterior communicating arteries.[14]

HAMBY'S[15] CASE.—A man aged 24 had recently undergone an army medical examination, when his vision was found to be normal. He suddenly experienced a severe headache. Three days later dazzling lights appeared, and this was followed by blindness. The headache continued. On examination it was noted that he apparently "would go out of his way to walk into furniture." Neurological examination showed nothing abnormal; the cerebrospinal fluid showed 186 leukocytes per cubic millimeter and 130 mg. of protein per 100 cc. On ventriculographic examination each cannula entered an abscess. These abscesses were metastatic and attributable to bronchiectasis.

I have seen a case in which a cardiac thrombus resulting from an infraction of the coronary artery gave rise to multiple cerebral embolism, so that homonymous hemianopsia was followed by sudden complete blindness in an interval of 14 days.

Vascular Lesions of Optic Nerves.—Retrobulbar vascular lesions involving the vasa nervorum of the optic nerves occasionally occur as a result of arteriosclerosis and arteritis and give rise to central scotoma or amaurosis in apoplectiform fashion. Polyarteritis of the type seen in association with temporal arteritis is particularly likely to give rise to unilateral or bilateral amaurosis fulminans in this manner.

Disseminated Demyelinating Diseases.—These are a common cause of amaurosis, and the process may involve the visual pathways at any point. For clinical reasons chiefly, they have been divided into four principal types: disseminated sclerosis, Devic's disease (neuroencephalomyelitis optica), Schilder's disease (progressive subcortical encephalopathy) and acute disseminated encephalomyelitis.

Many nerve fibers are covered by a layer of myelin of variable thickness. At some distance outside the brain stem and spinal cord the nerve fibers receive a further covering of reticular cytoplasm—the sheath of Schwann, or neurilema. Between two nodes of Ranvier this cytoplasmic layer comprises one cell, complete with nucleus. The nerve fibers within the optic nerves, however, do not receive this cover. Myelinated nerve fibers not protected by neurilema are liable to direct damage in any of the disseminated demyelinating diseases. Since the proximal segments of the cranial nerves remain devoid of neurilema, isolated paralyses of oculomotor and other cranial nerves are seen occasionally in disseminated sclerosis.

13. Hemphill, R. E.: Sudden Blindness of Cortical Origin as Result of Cerebral Vascular Lesion, Irish J. M. Sc. **181**:28, 1941.

14. Kubick, C. S., and Adams, R. D.: Occlusion of Basilar Artery: Clinical and Pathological Study, Brain **69**:97, 1946.

15. Hamby, W. B., in discussion on Riley, H. A.; Yaskin, J. C.; Riggs, M. E., and Torney, A. S.: Bilateral Blindness Due to Lesions in Both Occipital Lobes, New York State Med. **43**:1632, 1943.

The clinical and histological criteria are not sufficiently distinctive to permit clearcut nosological distinction between the various forms of disseminated demyelinating encephalomyelitis. There is a growing tendency to regard many of the acute forms as belonging to the same group as that which may be initiated by a number of exanthems (vaccinia, smallpox, chickenpox, measles, rubella and scarlet fever). A similar reaction may follow the Pasteur treatment for rabies, repeated subcutaneous injections of sterile brain tissue extracts into experimental animals and the exhibition of various therapeutic serums in man. This "allergic" form of encephalomyelitis has recently been the subject of several experimental studies, and certain conclusions have been drawn.[16] The active factor which induces this encephalomyelitis is absent from the frog and fish brain. It is unaffected by boiling or by treatment with formaldehyde and gives rise to a progressive encephalomyelitis in about 50 per cent of the animals after a varying interval.

Anoxia and a number of poisons which interfere with oxidative processes are known to give rise to widespread, and often delayed, disseminated encephalomyelitis.[17] The following case probably belongs to this group.[18]

There had been cardiac arrest during anesthesia for 5½ minutes. On the following day the patient, a boy aged 12, said he was well. On the third day he complained of dimness of vision; on the fourth day he could distinguish only between light and darkness and complained of "numbness" of the right hand. When I examined him on the fifth day, light perception was barely possible. Neurological examination revealed also signs indicative of multiple cerebral lesions. Both pupils reacted briskly to light, and the fundi appeared normal. Gradual recovery of vision occurred, so that when he was reexamined three weeks later visual acuity and fields were within normal limits and the neurological status was also normal.

A common form of acute disseminated encephalomyelitis is bilateral papillitis or retrobulbar neuritis. I have known permanent blindness to occur in a space of 24 hours. Occasionally onset of blindness may be preceded or followed by myelitis or encephalitis. A recent case referred by Mr. Shapland illustrates this well.

A child aged 3½ years was given tetanus antitoxin after she had been bitten by a dog. Three weeks later she became jaundiced, without showing any pronounced constitutional symptoms, and within a few days she was well again. Six weeks following the injection of the serum, she became blind in the space of a few hours. Examination on the following day revealed intense bilateral papillitis. During the subsequent week she recovered much of the vision, but one morning during the eighth week she appeared unusually drowsy. Shortly afterward she had an epileptic seizure, became comatose and, after another series of fits, died about 48 hours later.

Space-Occupying Lesions in Region of Sella Turcica.—Blindness due to tumors or aneurysms in the region of the sella turcica is, of course, commonplace. I shall consider only space-occupying lesions in this region which may be said to give rise to amaurosis fulminans. Hemorrhage into intrasellar tumors, whether showing malignant changes or not, is a rare but well recognized condition. Such a catastrophe may bring about an expansion laterally and cause compression of the

16. Lumsden, C. E.: Experimental "Allergic" Encephalomyelitis, Brain **72**:198, 1949.

17. Lumsden, C. E.: Cyanide Leucoencephalopathy in Rats and Observations on Vascular and Ferment Hypotheses of Demyelinating Diseases, J. Neurol., Neurosurg. & Psychiat. **13**:1, 1950.

18. Bailey, H.: Cardiac Massage for Impending Death Under Anaesthesia, Brit. M. J. **2**:84, 1941.

cranial nerves within the cavernous sinus.[19] On the other hand, swelling may take place principally in an upward direction, so that the chiasm is pressed on.[20] More frequently, structures in the cavernous sinus and the chiasm are affected simultaneously.[21] Since hemorrhage into an intrasellar tumor is associated with intense headache and bleeding into the subarachnoid space, it may be difficult, without resort to arteriography, to distinguish this condition from an expanding intracranial aneurysm with rupture.

Unilateral loss of vision as a result of intracranial aneurysm is a familiar occurence which does not call for comment here. However, aneurysms, because of their midline position or their size, may be large enough to compress the chiasm and give rise to almost simultaneous bilateral blindness. I have had one such case: A sudden, intense headache was followed by blindness; after a while vision in one nasal field returned.

An aneurysm of the basilar artery may compress the chiasm from behind, but a lesion of this nature may be expected to give rise to cortical blindness.[22] Yaskin has reported a case of cortical blindness of about two weeks' duration following a subarachnoid hemorrhage, presumably caused by an aneurysm of the basilar artery.[23] Though the anterior tip of the basilar artery is a common situation for aneurysms, such lesions seem to give rise to bilateral oculomotor paralyses rather than amaurosis.

Closed Head Injuries.—Such injuries are not an infrequent cause of permanent bilateral amaurosis. Usually the lesion involves the optic nerves or chiasm and has been produced in the manner already described. Rarely it is the result of contusion of the occipital lobes or of subdural hematoma in an unusually posterior position. Since the fall leading to the head injury may have been due to epilepsy, amaurosis supervening on epilepsy may give rise to diagnostic difficulties.

Basal Meningitis.—Acute purulent meningitis may rarely be followed by blindness.

A patient aged 47, after two days of severe headache, became delirious. When she recovered consciousness three days later, she was totally blind. The diagnosis of meningococcic infection was confirmed bacteriologically. She received a course of sulfonamide therapy, to which she did not show any intolerance. About one month later she recovered some vision in the central portion of the left field. Thrombosis of vessels of the chiasm and tracts is the explanation which suggests itself.

Chronic forms of basal meningitis have received a good deal of attention during the past 20 years, especially in the French literature, under the name "chronic chiasmal arachnoiditis." For reasons which are oftener than not obscure, the

19. Coxon, R. V.: A Case of Haemorrhage into a Pituitary Tumor Simulating Rupture of an Intracranial Aneurysm, Guy's Hosp. Rep. **92**:89, 1943.

20. Walsh, F. B.: Bilateral Total Ophthalmoplegia with Adenoma of the Pituitary Gland: Report of 2 Cases, An Anatomic Study, Arch. Ophth. **42**:646 (Nov.) 1949.

21. Benjamin, R. E.: Pituitary Tumor with Fulminating Symptoms, J. A. M. A. **92**: 1755-1758 (May 25) 1929.

22. Dandy, W. E.: Intracranial Arterial Aneurysms, New York, Comstock Publishing Co. Inc., 1947.

23. Yaskin, J. C., and Spaeth, E. B.: Acute Cortical Blindness with Recovery: Report of a Case, Arch. Neurol. & Psychiat. **54**:70 (July) 1945.

pia-arachnoid in this region may undergo proliferative inflammatory charges, and these may lead to a pronounced interference with the blood supply to the optic nerves and chiasm. Disturbances of vision often have an apoplectiform onset and may be subject to fluctuations and remissions. Such vascular episodes may be associated with progressive changes of more insidious nature. These clinical features occuring without pain should be regarded as suggestive of chronic chiasmatic arachnoiditis.

Embolism.—Recently I have had two cases of embolism, one of air and the other of oil embolism; in both cases disturbances of vision were prominent features.

Air Embolism: A woman aged 25 had had artificial pneumothorax (left side) two years previously. At the time of her visit the sputum was free from tubercle bacilli. At 7 p. m. she had a second injection of air. On walking out of the room, she suddenly collapsed and became pulseless. Both eyes were fully deviated to the right. She recovered consciousness about a quarter of an hour later and immediately complained of loss of vision. The neurological status was normal; the pupils reacted to light. The patient was virtually blind, light perception alone remaining. Her temperature rose to 101 F. She continued to vomit and had another fit two days later, after which she began to improve. Her vision became normal again on the fourth day, and she was quite fit when discharged on the seventh day. The visual fields and visual acuity have remained normal.

Oil Embolism: A young woman received an injection of an oily solution of procaine for fibrositis, during which she collapsed suddenly. She had complete amnesia for events which occurred during the following fortnight. When she again became oriented, she was aware of impairment of vision. This condition gradually improved. When I saw her for the first time, about two months after the accident, she had complete homonymous hemianopsia with sparing of central vision.

Space does not permit me to dwell on such well described causes of amaurosis as quinine intoxication, hysteria and anemia following hemorrhage.

I should like to refer to a condition which may be regarded as lying somewhere between amaurosis fugax and amaurosis fulminans. Such cases are not uncommon. The patient usually does not describe any blackout or appearance of a black curtain, the chief symptom being absence of vision. After a variable interval, gradual recovery takes place, and on subsequent examination the patient usually shows complete homonymous hemianopsia. In many cases the lesion appears to have been due to thrombosis of one calcarine artery. Despite a diligent search, I have not found these cases described in the literature; yet a careful inquiry will reveal this sequence of events in many cases presenting homonymous hemianopsia of sudden onset. In view of the frequency of this occurence, I do not think that transient occlusion of both cerebral arteries could be the explanation.

In 1936 Hines discovered that electrical stimulation of a small strip of precentral cortex evoked inhibition of an existing contraction of the skeletal muscles on the opposite side of the body.[24] Dusser de Barenne found that stimulation of this region with strychnine resulted in cessation of all electrical activity of the cortex. It was further discovered that this reaction was mediated not transcortically, but through a complex circuit involving the basal ganglions and the thalamus. Using this technic, Dusser de Barenne later found other similar areas, which are now known

24. Hines, M.: The Anterior Border of the Monkey's (Macaca Mulatta) Motor Cortex and the Production of Spasticity, Am. J. Physiol. **116**:76, 1936.

as suppressor strips.[25] It would appear that each suppressor area has its own projection system linking it with the basal ganglions. One such suppressor area is situated in close proximity to the striate visual cortex. Strychninization of this suppressor area in the macaque and the chimpanzee leads to cessation of electrical activity of the cortex. Simultaneous electroencephalographic records of various portions of the cerebral cortex reveal that this loss of electrical activity spreads slowly over both the ipsilateral and the contralateral hemispheres.[26] As I see it, ischemia of the occipital cortex is liable to activate temporarily the suppressor area, and this may account for the transient loss of vision in both visual fields. It would equally well explain loss of consciousness, which may be a feature in these cases.

33 Harley Street.

25. Dusser de Barenne, J. G., and McCulloch, W. S.: Suppression of Motor Response Obtained from Area 4 by Stimulation of Area 4s, J. Neurophysiol. **4**:311, 1941.

26. Bonin, G.; Garol, H. W., and McCulloch, W. S.: Functional Organization of the Occipital Lobe, Biol. Symposia **7**:165, 1942.

Clinical Notes

A REVERSIBLE BIFOCAL LENS

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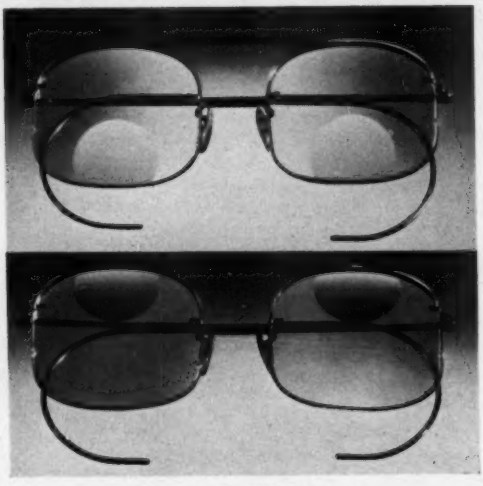
A METHOD for inverting a bifocal lens is described. A standard Bausch & Lomb "Bal-Grip" mounting is utilized. The lens itself is shaped like a symmetrically "rounded square." This pattern is available in "two difference," "four difference" and "six difference" optical sizes. The lower portion of the lens is notched so that the lens can be inverted when desired. Any type of bifocal or trifocal can be used. The accompanying illustration clearly shows the reversible bifocal. It consists of the regular "Bal-Grip" mounting with a bridge size of



The reversible bifocal lens.

22 mm., a temple length of 6.5 inches (16.5 cm.) and lens size of 46 by 42 mm. The illustrated bifocal is Tillyer B. The lens is shown in both positions.

TECHNIC OF FITTING THE LENS

The bridge size is selected and fitted. The near interpupillary distance is then measured. An eye size is arbitrarily selected so that the horizontal dimension of the lens, plus the size of the bridge, will be equal to the near interpupillary distance. This will maintain the bifocal segment in a position near the geometric center of the lens and, at the same time, center it for near vision. A procedure of this type instead of the usual method of bifocal decentration, must be followed to prevent the decentration "in" when the segment is below from developing into decentration "out" when the segment is above. The bifocal position is, therefore, set for near, and distance prismatic effects are omitted from consideration. This omission is not nearly as serious as it would seem at first. Individual patient tolerance to prism "base in" is well known. Prism "base out," which one frequently encounters in minus cor-

rections, should be more carefully watched. During the course of the examination, however, these tolerances can be measured and established, and only those patients who can tolerate the induced prismatic effect need be considered. These would include nearly all hypermetropic patients, together with the great majority of patients with low and medium myopia. The lens would not be applicable in cases of high myopia because the degree of "base-out" prism at distance would be too extreme. It might be noted at this time that the reversible bifocal lens is rotated, and not tilted. The rotation, of course, preserves the original cylinder axis. The lens is made in toric form, so that it has to be rotated to be reinserted properly into its mounting in either the upper or the lower position.

ADVANTAGES

This reversible lens arrangement presents certain advantages. Its flexibility will save many patients an extra pair of glasses. It offers a possible solution to the long-standing problem of the patient who cannot accustom himself to walking while wearing bifocals in their usual position. It also permits the bifocal segment to be placed upward and out of the way during all or part of his walking activities. It would also seem applicable for patients engaged in occupations with unusual visual demands. Patients in these categories include barbers, switchboard operators, painters and paper hangers, as well as most people employed in the building trades, where sure footing is a necessity.

This lens has several advantages also from the standpoint of the practitioner. Any general optical laboratory has the necessary tools and equipment to manufacture it. Its economy might be stressed, since the cost of the notches constitutes the only added labor item. The technic of prescribing and fitting this lens can be easily mastered by any one who has a basic knowledge of elementary optics. The mechanics of changing the position of the lens is sufficiently simple that any intelligent patient can master them easily. There are no limitations regarding the size, shape or type of bifocals that can be used, and this applies to trifocals as well. The "rounded square lens" was chosen for its mechanical advantages, rather than for its aesthetic properties. However, most patients are rather pleased with its appearance. The reversible bifocal lens can also be manufactured in octagonal and round forms, but for technical reasons and manufacturing simplicity the "rounded square" would seem to be preferable.

1017 Republic Bldg. (15).

COMMERCIALLY PREPARED PACK (THE HYDROCOLLATOR)

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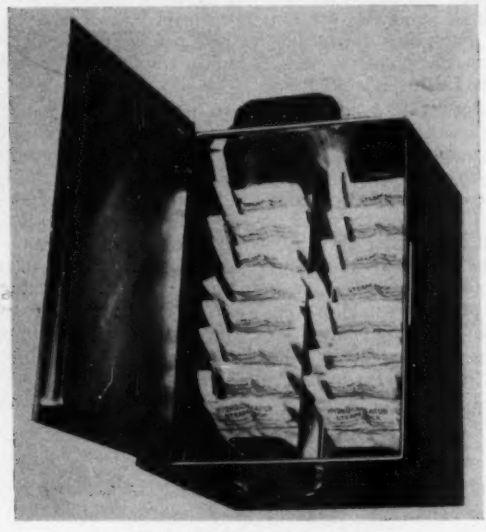
Moist heat is an extremely effective measure in treatment of inflammations of the eyelids and the anterior segment of the globe, and at first glance it would be hard to think of a simpler method than the application of hot moist pads. The application, to be of any value, however, must be continued for 15 minutes or more, during which time the pad must be kept moist and the temperature constant. In these days of personnel shortages, hospital nursing staffs, with the best will in the



The Hydrocollator steam pack.

world, find it difficult, and often impossible, to carry out orders for moist heat when it means tying up the time of a nurse or an attendant with one or two patients for a relatively long time while these requirements are met.

Some months ago there came to my attention a commercially prepared pack (the Hydrocollator steam pack), which retains moist heat for 30 minutes and which does away completely with the handling of dripping towels or gauze. It struck me that if such a pack could be prepared for ophthalmologic use the application of moist heat could be accomplished more effectively and the personnel difficulties to which I have just referred would be largely solved.

With the cooperation of the manufacturer, my colleagues and I began to use a similar ophthalmic pack at the Eye, Ear, Nose and Throat Hospital in New Orleans

Read before the Section on Ophthalmology at the Ninety-Ninth Annual Session of the American Medical Association, San Francisco, June 29, 1950.

last October. We eventually succeeded in devising a pack which has proved perfectly satisfactory for our purposes.

This pack is made from a silica gel which is so processed that it hydrates rapidly and completely and retains moisture and heat for as long as 30 minutes. Silica gel in an inert substance, so that any chemical or other reaction from its use is impossible. The pads are 2 inches (5 cm.) square and are stitched in a cruciate fashion, so that they are divided into inch squares and the contents cannot collect in any single portion. The tab at the corner permits ready handling, even when the pad is very hot. A specially devised heater permits 16 pads to be heated at one time. It is thermostatically controlled, to keep the water at a constant temperature, just below the boiling point, and to provide a supply of pads for use throughout the day.

When the pad is first removed from the heater, it is too hot to be applied directly to the lids. Instead, it is placed over two cotton pads, one of which is removed after five minutes, while the other remains in place for the remaining period of the treatment. These pads are 3 inches (7.5 cm.) square. They are autoclaved in pairs and are reusable after they have been washed and autoclaved again.

The advantages of this technic are obvious. The eye pads, as I have pointed out, are perfectly safe to use. Patients find them comfortable and soothing and like the idea of not being disturbed constantly, as was necessary when the old-fashioned gauze compresses were used. The initial cost of the pad is, of course, a considerable item, but it is compensated for by their long life; the larger pads from which these eye pads were modeled have been used, in some instances, for two years. The cotton pads, since they are reusable, cost almost nothing. The greatest therapeutic advantage of the method, however, is the more effective application of moist heat, while the greatest practical advantage is the more efficient use of hospital personnel. If the applications in a single ward, or in a series of wards, are synchronized, as they can easily be, one nurse or other attendant can care for 10 or 12 patients within a period of one half to three quarters of an hour.

Our experience with this method at the Eye, Ear, Nose and Throat Hospital has been highly satisfactory in every respect, and I have no hesitancy in recommending this technic for general use.

Annual Reviews

LENS AND VITREOUS

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WITHIN the last year many reports have appeared evidencing noteworthy progress in the investigations of the fundamental problems of the composition and metabolism of the lens and vitreous humor. Efforts were made to solve the problems of the etiology, prophylaxis and treatment of retrolental fibroplasia. The addition of hyaluronidase to procaine solution and the use of curare constituted important advances in the technic of anesthesia for cataract surgery. Publications have appeared on the use of "physiologic glue" for closure of corneal sections. Because of the mounting tension between the Western Nations and those of the Soviet bloc, effects of atomic energy radiations are assuming an ever increasing importance, and concurrently reports of clinical and experimental investigations of various types of radiation cataract have appeared.

CHEMISTRY AND METABOLISM

Respiration.—Merriam and Kinsey [1] have made a valuable contribution to a more intensive study of lens metabolism by developing an apparatus for in vitro lens culture. Their method, termed the "batch" technique, has the advantage over the formerly used perfusion technique that it is possible to demonstrate significant quantitative changes in the concentrations of the lens metabolites in the fluid bathing the lens. These authors suspended lenses in four different mediums, two of which were synthesized to approximate the composition of the aqueous, while the third was supplemented with amino acid concentrate and the fourth consisted of beef aqueous humor to which dextrose had been added. In general, over a period of days the glucose uptake and the lactic acid production decreased, indicating a progressive loss in the lens vitality. The investigators determined the glutathione and ascorbic acid content of all the lenses. They found that in every lens in which there was the slightest loss of transparency there was a decrease in glutathione content. To quote the authors:

> These changes in metabolism appear to represent more sensitive indexes of lens injury than does the physical appearance, and suggest that loss of transparency may be a relatively late secondary effect of metabolic changes.

Thus, to the growing list of cataracts in which decreased glutathione has been reported prior to the appearance of opacities, we may now add opacities appearing in explanted lenses. Unfortunately, the value of these in vitro experiments may be limited, for Langham and Davson [2] have proved that degenerative changes occur speedily in excised lens, as indicated by the rapid uptake of Cl and Na ions.

From the Department of Ophthalmology, Northwestern University Medical School.

1. Merriam, F. C., and Kinsey, V. E.: Studies on the Crystalline Lens: I. Technic for in Vitro Culture of Crystalline Lenses and Observations on Metabolism of the Lens, Arch. Ophth. **43**:979-988 (June) 1950.

2. Langham, M., and Davson, H.: Studies on the Lens, Biochem. J. **44**:467-470, 1949.

Merriam and Kinsey [1] report that the minimal loss of glutathione occurred in those lenses suspended in the solution containing amino acid. The explanation for this finding may lie in the ability of the lens to synthesize glutathione from amino acids. This observation is substantiated in a second paper by the same authors,[2] in which they report on the measurement, by means of radioactive glycine, of the synthesis of glutathione in the normal and the cataractous rabbit lens. They showed that the glycine molecule is incorporated into the glutathione as a whole rather than being broken down to carbon dioxide and then resynthesized. From a study of the rate of turnover in the tissues of labeled glycine in glutathione, it appears that, although in cataractous lenses there may be some increase in the rate of degradation of glutathione, the primary effect is an interference of resynthesis of the glutathione. This finding is in accord with the newer concepts of energy relationship in the body in that distinction of exogenous and endogenous metabolism has been abolished.

Ely [4] investigated the oxygen uptake of various portions of the lens. He found that the entire oxygen uptake is explained by activity on the part of the epithelium and the cortical fibers. He also showed that for all animals studied the growth curves and percentage water contents of comparable age groups are similar.

Proteins.—Mandel and his group [5] report the presence of 8 mg. of phosphoprotein per 100 Gm. of fresh lens tissue. They found that the nucleic acid is apparently in the ribonucleic fraction of the lens. The concentration of this substance varies from 33 to 36 and from 42 to 47 mg. per 100 Gm. of fresh tissue in the sheep and the ox, respectively.

Amino Acids.—Schaeffer and Murray [6] have carefully analyzed the amino acid content of the beef lens protein. The concentrations of lysine, histidine and arginine are very similar to those found earlier by Krause, although appreciably different values for tyrosine, cystine and tryptophan are reported. In another report Schaeffer and Murray [7] demonstrate that the essential amino acids are necessary for the proper growth of the lens, as well as the body. The absence of any essential amino acid in the diet may result in the formation of cataract. A delayed supplementation of a tryptophan-deficient diet does not prevent the formation of cataract. It is evident that all the essential amino acids must be present in the diet simultaneously for the normal growth and development of the lens. The authors showed that when tryptophan is absent from the diet, or is administered independ-

3. Kinsey, V. E., and Merriam, F. C.: Studies on the Ocular Lens: II. Synthesis of Glutathione in the Normal and Cataractous Rabbit Lens, Arch. Ophth. **44**:370-380 (Sept.) 1950.

4. Ely, L. O.: Metabolism of the Crystalline Lens: I. Water Content and Growth Rate, Am. J. Ophth. **32**:215-219 (June, pt. 2) 1949; II. Respiration of the Intact Lens and Its Separated Parts, ibid. **32**:220-224 (June, pt. 2) 1949.

5. Mandel, P.; Nordmann, J.; Zimmer, J., and Harth, S.: A Phosphoprotein in the Lens of the Eye, Nature, London **164**:794, 1949. Mandel, P.; Nordmann, J., and Zimmer, J.: Acide nucleique du cristallin, Compt. rend. acad. sc. **288**:516, 1949.

6. Schaeffer, A. J., and Murray, J. D.: Amino Acid Composition of the Lens Proteins of the Bovine Eye, Arch. Ophth. **43**:1056-1064 (June) 1950.

7. Schaeffer, A. J., and Murray, J. D.: Tryptophan Determination in Cataracts Due to Deficiency of Delayed Supplementation of Tryptophan, Arch. Ophth. **43**:202-216 (Feb.) 1950.

ently of the other amino acids (delayed supplementation), protein synthesis becomes impossible. These findings prove that tryptophan deficiency cataract is really a protein deficiency cataract.

Vitamin C.—Kinsey [8] proved that the ascorbic acid in the aqueous humor is not synthesized by the lens, a hypothesis formulated earlier by Fischer. He also throws grave doubt on the theory that ascorbic acid enters the aqueous in the form of dehydroascorbic acid and is then reduced to ascorbic acid by the lens. He believes that ascorbic acid itself is secreted by the ciliary body and reaches the anterior chamber with some loss. The reason for the marked diminution in the content of the aphakic eye remains unsolved.

Cytochrome C.—It has never been unequivocably shown that cytochrome C exists in the lens, though its presence has been intimated by Herrmann and Moses. Ely and Robbie [9] have shown that an enzyme system which can be inhibited by cyanide is present in the lens. However, they were unable to prove that this system is cytochrome C, and they concluded that either it is absent from the lens or it is present in quantities of less than 20 micrograms per gram. The mechanism of the breakdown of lactic acid to carbon dioxide and water remains unsolved.

Fluorescence.—Brolin and Krakau [10] investigated the fluorescence in various spectral ranges of the lens in cases of galactose and naphthalene cataract. They found that the maximal changes in fluorescence occurred at about 490 microns and that various types of cataract showed increases over the normal at this wavelength.

Hydrogen Ion Concentration.—De Vincentiis [11] studied the effects of hydrogen ion concentration on the ectoderm forming the crystalline lens in Rana esculenta. He found that variations in the pH prevents the in vitro development of the lens.

CONGENITAL ANOMALIES

Congenital Cataract.—That cataract occurs in a hereditary form has been known for over two centuries. The descent is preponderantly dominant, although irregular dominance and recessivity occur. Sæbø [12] examined 17 families in which 27 cases of congenital and juvenile cataract occurred, 14 members being males and 13 females. Twenty cataracts were considered to be congenital and seven juvenile in type. In five of the 17 families the parents of the affected persons were first cousins, and in four more they were related, although the relationship was more distant.

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8. Kinsey, V. E.: Dehydroascorbic Acid in the Aqueous Humour of Rabbits, Am. J. Ophth. **33**:257-266, 1950.

9. Ely, L. O., and Robbie, W. A.: The Cyanide Sensitivity and Cytochrome-C Content of the Crystalline Lens, Am. J. Ophth. **33**:269-272, 1950.

10. Brolin, S. E., and Krakau, T.: Spectrophotometric Investigations on the Fluorescence of the Ocular Lens with Changed Metabolism in Rats with Galactose, Acta ophth. **27**:291-301, 1949. Brolin, S. E.: Spectrophotometric Investigations on the Fluorescence of the Eye Lens in Rats Given Naphthalin, ibid. **28**:163-177, 1950.

11. de Vincentiis, M.: Ricerche sperimentali negli anfibi (*rana esculenta*) sulla embriogenesi del cristallino in condizioni di espianto, Rassegna ital. d'ottal. **18**:217-236, 1949.

12. Sæbø, J.: An Investigation into the Mode of Heredity of Congenital and Juvenile Cataracts, Brit. J. Ophth. **33**:601-629, 1949.

Marner [13] describes a Danish family in which cataract occurred over a period of eight generations. In 1878 Giersing reported that 20 of 25 members of this family had cataract, and in 1896 Norrie found 40 cases of cataract in 69 members. Today the family has 542 members, of which 132, or 24 per cent, have cataract. The cataract descent is dominant, with cataract developing in 50 per cent of the descendants of affected members. The occurrence as to sex is almost equally divided, 47 per cent occurring in males and 53 per cent in females. The author believes that the incidence in this family was probably higher than reported because there were undoubtedly some cataracts among the 21 members who died in infancy. Other hereditary defects were absent. The cataracts in all cases were bilateral, and the morphology was similar in the majority of cases. The cataracts were progressive in character, being of slow development in most instances, although more rapid in others. The fact that the cataracts are now appearing at an earlier age makes it necessary for children of school age to undergo operation. The postoperative results are incomplete, but, from the material available, visual acuity seems to depend on the age when operation was performed. The visual acuity of children requiring operation at an early age was less than that of children in whom surgical intervention was not necessary until after the eighth or ninth year. This difference in visual results in the very young and in the older group was attributed to a greater degree of amblyopia in the younger group.

Redl [14] publishes a pedigree of six generations with cataract. In the latter three generations the cataract appeared as a congenital or early acquired perinuclear opacity. This observation raises the question of whether mixed forms of cataract may not exist on the basis of the same gene.

Smelser and von Sallmann [15] describe the morphological changes in hereditary cataract developing in a strain of Swiss albino mice. A thinning of the lens capsule and a slight disorganization of the lens fibers at the posterior pole were observed early. There was a diminution in the number of lens epithelial cells per unit area in the early stages of the cataract. After vacuolation of the lens and rupture of the capsule, the nucleus was extruded into the vitreous. An electrophoretic analysis of normal mouse lens proteins disclosed three major components, which, however, were not identified. One of these components was reduced at a rate almost twice as fast as that of the other two during the cataract formation.

Basing his opinion on the manner in which the lens develops, Winkler [16] states that a fusiform type of congenital cataract does not exist. He stresses the fact that most publications dealing with this type of opacity appeared prior to the introduction of the slit lamp.

The pathogenesis of anterior polar cataract is still obscure. The following factors have been credited in producing such opacities: (1) the absence of the protecting influence of the iris on the central portion of the lens capsule, (2) the merging of the anterior sutural lines and (3) the anomalies of circulation which

13. Marner, E.: A Family with 8 Generations of Hereditary Cataract, Acta ophth. **27**:537-551, 1949.

14. Redl, T.: Über eine Sippe mit teils präseniler, teils infantiler erblicher Katarakt, Klin. Monatsbl. Augenh. **114**:24-32, 1949.

15. Smelser, G. K., and von Sallmann, L.: Correlation of Microscopic and Slitlamp Examinations of Developing Hereditary Cataracts in Mice, Am. J. Ophth. **32**:1703-1712, 1949.

16. Winkler, G.: Gibt es einer Spindelstar? Klin. Monatsbl. Augenh. **115**:46-51, 1949.

may result from failure of the iridopupillary membrane to resorb. Substantiating the last theory, Waardenburg [17] has found evidence of a delayed resorption of membrane rests, which are strongly adherent to the center of the lens. According to this author, the degree of resorption of the iridopupillary membrane is dependent on hereditary factors, for in the family he described the entire syndrome behaved like a mendelian dominant with a great variability of expression.

The combination of interstitial keratitis, syphilitic choroiditis and embryonal cataracts is rare, according to Rosen.[18] In the case he cites, the opacities were located in specific zones of the lens, indicating a metabolic disturbance during the period of the syphilitic inflammatory reaction.

Ectopia Lentis.—An extensive new pedigree of Marfan's disease is described by Lutman and Neel.[19] In a kinship of 40 persons, 17 had Marfan's syndrome in varying degree of completeness. In addition to ectopia lentis or coloboma of the lens, the authors noted anomalies of the zonular fibers, iris and uveal pigment. It appeared that the syndrome was caused by a single gene, influenced by other genetic and environmental factors. The fact that the sporadic form of the disease is apt to be severer than the inherited form adds support to the mutation hypothesis of the origin of this disease.

Callahan [20] reports his observations on a family showing aniridia, ectopia lentis and glaucoma. The trait appeared as a dominant characteristic. The ectopic lenses were consistently found to be at least partially opaque. Judging from the visual fields, the eyes of these patients seemed to withstand the effect of increased intraocular tension better than the eyes of those with ordinary glaucoma.

Lenticonus.—Deformities of the lens appear rarely. Doherty [21] reports one case. After reviewing the literature, he classifies the deformities of the lens as follows: (1) true deformities, which include lentiglobus, lenticonus anterior and lenticonus posterior, and (2) false abortive forms, which include lenticonus internus, senile or acquired lenticonus and umbilication of the lens.

Fronimopoulos and Joannidis [22] describe a case of a young man with anterior lenticonus and rudimentary posterior lenticonus. Apparently in their case the condition was acquired, because the patient had good vision up to the age of 20 years.

RETROLENTAL FIBROPLASIA

Retrolental fibroplasia, a disease which was little known before 1940, is now recognized as the most important cause of blindness in the very young. This disease, usually appearing after the first postnatal month, occurs in premature

17. Waardenburg, P. J.: Gross Remnants of the Pupillary Membrane, Anterior Polar Cataract and Microcornea in a Mother and Her Children, Ophthalmologica **118**:828-842, 1949.

18. Rosen, E.: Embryonal Cataract Associated with Interstitial Keratitis and Syphilitic Choroiditis: Report of a Case, Arch. Ophth. **42**:749-754 (Dec.) 1949.

19. Lutman, F. C., and Neel, J. V.: Inheritance of Arachnodactyly, Ectopia Lentis and Other Congenital Anomalies (Marfan's Syndrome) in the E. Family, Arch. Ophth. **41**:276-305 (March) 1949.

20. Callahan, A.: Aniridia with Ectopia Lentis and Secondary Glaucoma: Genetic, Pathologic, and Surgical Considerations, Am. J. Ophth. **32**:28-40 (June pt. 2) 1949.

21. Doherty, W. B.: A Discussion of Deformities of the Shape of the Lens, with a Report of a Case of Posterior Lenticonus, Am. J. Ophth. **31**:457-460, 1948.

22. Fronimopoulos, J., and Joannidis, T.: Bilateral Anterior and Rudimentary Posterior Lenticonus, Arch. Ophth. **43**:864-870 (May) 1950.

infants and is bilateral. In the fully developed stage of this disease the eyes are abnormally small, the anterior chamber is shallow and a gray membrane of varying thickness lies behind the lens. Glaucoma is a frequent complication.

The cause of retrolental fibroplasia is unknown. The incidence appears to vary in different parts of this country. The disease was practically unknown in Europe, but recently cases have been reported, and they appear to be on the increase. Thus, Holm [23] states that his is the first case in Denmark. Moffatt [24] found that among 119 children in institutions for the blind in England there were 12 cases of retrolental fibroplasia and two cases in which that diagnosis was probable. Crosse [25] reports that in Birmingham, England, there were four cases of retrolental fibroplasia among 4,000 premature infants who were followed to the age of 1 year. In France, Lelong and associates [26] report two cases of retrolental fibroplasia and state that, in their opinion, the disease is inflammatory in nature and probably infectious in origin. All writers agree that prematurity is a factor. Owens and Owens [27] report that it occurred in 16 per cent of premature infants weighing less than 3 pounds (1,307 Gm.) at birth and in 8 per cent of those weighing between 3 and 4 pounds (1,307 and 1,814 Gm.) at birth.

The chief point to be determined in the etiology of this disease is whether it is congenital or acquired. If the disease is congenital, the investigators should direct their attention to maternal causes, such as infections and metabolic disease. On the other hand, if the disease is acquired, the investigations should be limited to the study of environmental and metabolic factors in the premature infant. Reese and his co-workers,[28] and also Klien,[29] believe that the disease is congenital, the matrix being present at birth and consisting of hemangiomas situated at the base of the vitreous.

The Owens contend that the disease develops after birth and that the eyes are entirely normal at the time of birth. Unsworth's [30] observations support these findings. After an extensive investigation, Heath [31] came to the conclusion that retrolental fibroplasia is a primary retinal disease with secondary retinal detachment.

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23. Holm, E.: Retinopathia Præmaturorum (Retrolental Fibroplasia), Acta ophth. **27**:623-631, 1949.

24. Moffatt, P. McG.: Clinical Aspects of Retrolental Fibroplasia, Proc. Roy. Soc. Med. **43**:223-227, 1950.

25. Crosse, V. M.: Retrolental Fibroplasia, Proc. Roy. Soc. Med. **43**:232, 1950.

26. Lelong, M.; Velter, E.; Desvignes, P.; Rossier, A., and Baron, J. B.: Deux cas de fibroplasie rétrolentale, Bull. Soc. d'opht. de France, 1950, pp. 73-77.

27. Owens, W. C., and Owens, E. U.: Retrolental Fibroplasia, Am. J. Pub. Health **40**:405-408, 1950; Retrolental Fibroplasia in Premature Infants, Am. J. Ophth. **32**:1-18, 1949; Retrolental Fibroplasia in Premature Infants: II. Studies on the Prophylaxis of the Disease: The Use of Alpha Tocopherol Acetate, ibid. **32**:1631-1637, 1949.

28. (*a*) Reese, A. B.: Persistence and Hyperplasia of Primary Vitreous: Retrolental Fibroplasia—Two Entities, Arch. Ophth. **41**:527-552 (May) 1949. (*b*) Reese, A. B., and Blodi, F. C.: Retrolental Fibroplasia, Klin. Monatsbl. Augenh. **114**:18-24, 1949. (*c*) Speert, H.; Blodi, F. C., and Reese, A. B.: Retrolental Fibroplasia: A Hazard of Premature Birth, Am. J. Obst. & Gynec. **59**:246-258, 1950.

29. Klien, B.: Histopathologic Aspects of Retrolental Fibroplasia, Arch. Ophth. **41**:553-561 (May) 1949.

30. Unsworth, A. C.: Retrolental Fibroplasia or Ophthalmic Dysplasia of Premature Infants, Tr. Am. Ophth. Soc. **47**:738-771, 1949.

31. Heath, P.: Retrolental Fibroplasia as a Syndrome—Pathogenesis and Classification, Arch. Ophth. **44**:245-274 (Aug.) 1950.

In a group of cases presented by Gilger [32] the disease affected boys preponderantly. However, Speert, Blodi and Reese [28c] found that the abnormality occurs equally in the two sexes. Hepner, Krause and Davis [33] state that retrolental fibroplasia developed in four of five premature infants, who were shielded from light for 35 to 60 days after birth, thus proving that light was not an etiological factor in the production of this disease. King [34] believes that the incidence of multiple births is important only so far as multiple births are more apt to occur prematurely than at term. He found that maternal diseases during pregnancy bore no relation to the occurrence of retrolental fibroplasia and that the incidence of hemangiomas of the skin or mental retardation was no higher in the premature infants in whom retrolental fibroplasia developed than in the premature infants in whom it did not.

The authors who maintain that the disease is congenital give the following arguments: 1. The eye is smaller than it would be if the growth had ceased at some time after birth. 2. A true pupillary membrane arises from the collaret of the iris in some cases. 3. In 20 per cent of cases the disease is associated with hemangiomas of the skin. 4. In cases of fraternal twins the disease has not been observed in both twins. It would seem reasonable to expect that both infants would have the involvement if the disease were caused by an environmental or metabolic factor. 5. A vascular matrix was observed by Heath in the eye of a premature infant enucleated 18 hours after birth, and Reese describes it in one case.

The investigators who maintain that the disease is acquired present the following arguments: 1. The eyes are of normal size until the disease interferes with growth and atrophy ensues (the Owens, Unsworth, Heath). 2. The eyes of premature infants in whom the condition develops and the eyes of those in whom it does not are equally free from objective evidence of disease immediately after birth (the Owens, Unsworth). 3. The incidence of hemangiomas of the skin is no greater in premature infants who have the disease than in those who do not (King [34]). 4. The retrolental fibroplasia is of inflammatory origin and is caused by toxic effects of the subretinal fluid (Wolff [35]), a physicochemical factor (Unsworth) or infection (Lelong and associates). 5. Some unknown metabolic or nutritional disturbance is present and may be caused by harmful effects of certain mineral and vitamin preparations (Kinsey and Zacharias [36]). Further evidence is the prophylactic effect of alpha tocopherol (the Owens [37]).

Medical and surgical treatment of retrolental fibroplasia is unsatisfactory. Paul [37] used roentgen radiation in nine cases of retrolental fibroplasia and reports slight improvement in three and no improvement in six. The Owens report that

32. Gilger, A. P.: Retrolental Fibroplasia in Prematurely Born Children, Am. J. Ophth. **32**:917-929, 1949.

33. Hepner, W. R.; Krause, A. C., and Davis, M. E.: Retrolental Fibroplasia and Light, Pediatrics **3**:824-828, 1949.

34. King, M. J.: Retrolental Fibroplasia: A Clinical Study of 238 Cases, Arch. Ophth. **43**:694-711 (April) 1950.

35. Wolff, E.: Pathological Aspects of Retrolental Fibroplasia, Proc. Roy. Soc. Med. **43**:227-230, 1950.

36. Kinsey, V. E., and Zacharias, L.: Retrolental Fibroplasia: Incidence in Different Localities in Recent Years and a Correlation of the Incidence with Treatment Given the Infants, J. A. M. A. **139**:572-578 (Feb. 26) 1949.

37. Paul, T. O.: Blood Groups and Effects of Roentgen Irradiation in Retrolental Fibroplasia, Arch. Ophth. **41**:659-666 (June) 1949.

the results obtained with the prophylactic administration of alpha tocopherol acetate were encouraging. The course of the disease itself remains uninfluenced by tocopherol therapy.

CATARACTS ASSOCIATED WITH ENDOCRINE DISTURBANCES

The secretions from the endocrine glands play an important part in the metabolic processes. It is to be expected that dysfunction of certain endocrine glands may result in cataract. There is excellent experimental foundation for the belief that diabetes and parathyroid tetany are caused by endocrine dysfunction. Although little experimental evidence exists for the endocrine origin of dermatogenous cataract and myotonic dystrophy, clinical experience shows that endocrine dysfunction is sometimes present in these conditions, and therefore they are considered in this section.

Diabetic Cataract.—Little new material on diabetic cataract has appeared in the literature within the last year. Gordon [38] reviews the theories of the lenticular alterations that occur in diabetes. Gartner,[39] in an excellent article on the ocular pathology of diabetes, considers the changes in the lens. Roberts' [40] report stresses the reversibility of lens opacities in some cases of diabetes mellitus.

When the fundus is invisible because of diabetic cataract, the Rumpel-Leede test for estimating the capillary fragility is a valuable aid in predicting the visual outcome of cataract extraction. The test is made by maintaining pressure on the upper arm for five minutes with a blood pressure cuff. A positive test, evidenced by petechiae, indicates the probable presence of vascular lesions in the retina. Sédan and his group [41] report that in patients with a positive Rumpel-Leede test the visual acuity was 0.1, while the visual acuity averaged between 0.5 and 0.6 in those for whom the test was negative.

Alloxan Cataract.—Although changes in the lens in diabetes were reported more than 150 years ago, the mechanism of cataract formation in diabetes is still unexplained. Many investigators have availed themselves of the new methods of producing diabetes experimentally in order to study this problem more thoroughly. The method of administration of alloxan, which is the most recent means of producing experimental diabetic cataract, was discovered by Dunn, Sheehan and McLetchie.[42] Bellows and Shoch [43] investigated the effect of alloxan in diabetogenic doses on the glutathione content of the rabbit lens and, in addition, with the aid of the ophthalmoscope and slit lamp, observed the progress of alloxan cataract. Within a short time after the onset of hyperglycemia all the rabbits showed numerous minute vacuoles in the extreme periphery of the lens. The earliest change in the glutathione

38. Gordon, B. L.: Effects of Diabetes on Cataract and on Vision, Arch. Ophth. **41**:462-472 (April) 1949.

39. Gartner, S.: Ocular Pathology of Diabetes, Am. J. Ophth. **33**:727-740, 1950.

40. Roberts, W.: Rapid Lens Changes in Diabetes Mellitus, Am. J. Ophth. **33**:1283-1285, 1950.

41. Sédan, J.; Levrat, M., and Paufique, L.: Le signe du lacet envisagé comme test d'opérabilité de la cataracte diabétique, Ann. d'ocul. **182**:177-187, 1949.

42. Dunn, J. S.; Sheehan, H. L., and McLetchie, N. G. B.: Necrosis of Islets of Langerhans Produced Experimentally, Lancet **1**:484-487, 1943.

43. Bellows, J. G., and Shoch, D. E.: Alloxan Diabetes and the Lens, Am. J. Ophth. **33**:1555-1564, 1950.

concentration occurred 72 hours after the injection of alloxan. At this time, a decrease of 40 to 60 per cent in the glutathione content appeared, provided a severe diabetes existed. If the injection of alloxan did not result in diabetes, there was no decrease in the glutathione and no visible alterations in the lens. Waters [44] reports that the total sulfhydryl (SH) content of the lens decreased immediately after injection of alloxan and the value remained low permanently. He demonstrated that the decrease occurs in the SH groups of the lens proteins.

Parathyroid Deficiency.—Parathyroid deficiency, either operative or spontaneous in origin, results in tetany and possible cataract. Lyle [45] reports a case in which tetany and cataract occurred as a result of an idiopathic deficiency. In his discussion of idiopathic hypoparathyroidism, Lyle describes the increased demands on the body made by pregnancy, lactation and menstruation and recommends measures for the prevention and treatment of the latent and manifest stages of this disease. Hagedoorn [46] describes four cases of idiopathic hypoparathyroidism, cataracts being present in three of these. In all, the tendon reflexes were absent or sluggish, a finding which is unusual in the presence of a low blood calcium.

Dystrophia Myotonica.—Cataract is the most consistent extramuscular symptom in dystrophia myotonica. Farber,[47] in his report, states that it is the most important of the anticipatory signs. The lens opacities appear at an earlier age in succeeding generations. Three cases are reported by the author. In two cases, in which the cataracts were observed in the incipient stage, the alterations were similar to those described by earlier writers, while in the third case the cataract was so far advanced that it could not be distinguished from ordinary senile cataract.

Cataract Associated with Mongolism.—Galindez [48] observed several types of cataract in patients with mongolism. The cataracts in this condition resembled those found in myotonic dystrophy. Multiple punctate opacities were common; at other times the opacities were shaped like disks, rings or flakes. The opacities were located in the deep cortex, and a clear space separated them from the lens capsule.

Dermatogenous Cataract.—The dermatological diseases producing cataract are usually placed in one of three categories: (1) neurodermatitis (atopy, prurigo diathesique); (2) scleroderma (Werner's syndrome), and (3) poikiloderma vasculare atrophicans (Rothmund's syndrome).

Neurodermatitis.—Cowan and Klauder [49] report eight cases of cataract among 100 patients with atopic dermatitis. The average age of the patients with cataract was 23 years. The cataract appeared first in the subcapsular region near the posterior pole and consisted of globules, vacuoles and fine punctate opacities, which multiplied, enlarged and finally coalesced, becoming whitish yellow. The opacities

44. Waters, J. W.: Biochemical and Clinical Changes in the Rabbit Lens During Alloxan Diabetes, Biochem. J. **46**:575-578, 1950.

45. Lyle, D. J.: Cataract and Tetany Produced by Parathyroid Deficiency During Pregnancy, Lactation and Menstruation, Am. J. Ophth. **32**:1183-1188, 1949.

46. Hagedoorn, A.: Idiopathische hypoparathyreoidie, cataract en niet op te wekken of lage peesreflexen, Nederl. tijdschr. v. geneesk. **51**:4281-4284, 1949.

47. Farber, D. N.: Cataract in Dystrophia Myotonica, Arch. Ophth. **41**:450-459 (April) 1949.

48. Galindez, I. F.: Catarata mongolica, Acta pediát. españ. **78**:891-893, 1949.

49. Cowan, A., and Klauder, J. V.: Frequency of Occurrence of Cataract in Atopic Dermatitis, Arch. Ophth. **43**:759-768 (April) 1950.

then extended forward and peripherally, while similar changes occurred in the anterior subcapsular zone. At times a plaquelike opacity with wrinkling of the capsule developed. During the period from 1934 to 1948 Katavisto [50] observed 58 cases of prurigo diathesique, in 12 of which cataract developed. The average age at which the lens opacities appeared was 27 years.

Rothmund and Werner Syndromes.—The Rothmund-Werner syndrome was found by Jorio [51] in two cases and in one the disease was associated with Sjögren's syndrome. The author considers that a dysfunction of the sex glands plays an essential role in the etiology of this condition. Werner's syndrome is considered by Schott and Dann [52] to be an abiotrophic process which manifests itself by retarded growth during adolescence and by early graying of the hair. The changes in the skin are produced by atrophy of the subcutaneous fat and muscle. The lenticular opacities develop at the posterior pole and in the periphery.

Important articles are published by Franceschetti and Maeder [53] concerning cataracts associated with poikiloderma and scleroderma. They found that poikiloderma vasculare atrophicans (Rothmund's syndrome) occurred more commonly in the female. The heredity appeared recessive. The disease was characterized by poikiloderma of the Zinsser type and occurred in the early years of life. Pigmentary disturbances, atrophy and telangiectasias of the face, ears, forearms, hands and legs were present. Endocrine disturbances of the sexual type were found, with amenorrhea in the female and atrophy of the testes in the male. Mentally the patients were normal. Cataracts appeared between the second and the sixth year of age and progressed rapidly. Franceschetti and Maeder report that scleroderma, or Werner's syndrome, is a progressive disease and is characterized by diffuse scleroderma, involving particularly the lower limbs, and in a less degree, the forearms, hands and face. Pigmentary changes and telangiectasias were also found in their patients, making it difficult to differentiate this disease from Rothmund's syndrome. Later, ulcers of the legs and hyperkeratosis of the hands and feet were usually present. The appearance of senility developed early in life. Endocrine disturbances similar to those encountered in Rothmund's disease were usually present. The dermatological disturbances and the cataract developed later (at the age of 15 to 35) than in cases of the Rothmund type. Hereditary transmission was effected by irregular dominance with anticipation. The sexes were equally involved.

CHANGES IN THE LENS WITH AGE, MYOPIA, INFECTIONS, TOXINS, TRAUMA AND GLAUCOMA

Changes in the Lens with Age and Myopia.—The incidence of cortical cataract increases constantly and rapidly from the fortieth to the eightieth year. In Düsseldorf, Pau,[54] in tabulating the number of patients undergoing cataract extraction,

50. Katavisto, M.: Prurigo diathésique Besnier and cataract, Acta ophth. **27**:581-589, 1949.

51. Jorio, S.: Su due casi di cataratte simdermatotiche, Rassegna ital. d'ottal. **18**:17-33, 1949.

52. Schott, J., and Dann, S.: Werner's Syndrome: A Report of 2 Cases, New England J. Med. **240**:641-644, 1949.

53. Franceschetti, A., and Maeder, G.: Cataracte et affections cutanées du type poïkilodermia (syndrome de Rothmund) et du type sclérodermie (syndrome de Werner), Ophthalmologica **117**:196-198, 1949. Maeder, G.: Le syndrome de Rothmund et le syndrome de Werner: Étude clinique et diagnostique, Ann. d'ocul. **182**:809-854, 1949.

54. Pau, H.: Über Abnahme der Starmorbidität in Senium, Klin. Monatsbl. Augenh. **115**:53-55, 1949.

found that the percentage of cortical cataracts extracted decreased with age, especially in the 79 to 89 year group. It is to be expected that as nuclear sclerosis involves more and more of the lens, very little cortex remains to become opaque. It is for this reason that cortical cataract becomes less frequent in the group past the eightieth year. Since nuclear cataract usually allows sufficient vision for the very old, operation is ordinarily not considered necessary.

In another report, Pau [55] emphasizes the similarity of changes in the lens produced by age and those caused by myopia. Early nuclear pigmentation without myopia was interpreted as evidence of presenility. An increase in the size of the nucleus and early pigmentation were often associated with progressive axial myopia.

Cataracts Associated with Infections.—Punctate lenticular opacities are commonly found in adults. Legrand [56] examined 37 patients with this type of cataract and found that 30 had tuberculosis or a history of tuberculosis in their family. He discusses the toxic reactions to tuberculosis and the metabolic disturbances associated with this disease. Defective calcium metabolism is considered the chief factor in producing opacities of the lens. Auricchio [57] investigated the chemical and chemicophysical alterations in the aqueous humor and lens induced by tuberculous iridocyclitis in rabbits. He found that in this type of experimental iridocyclitis there is a reduction in the glucose content of the aqueous and lens, along with a diminished consumption of glucose by the lens fibers. An increase in the lactic acid content of the lens was also observed. The capsular permeability was at first increased and then reduced.

In an interesting article on cancer research, Mann [58] states that since both the cornea and the lens are free from malignant disease, it may be that the immunity depends on their avascularity, so that certain agents, possibly cancer-forming viruses, are unable to reach them.

Cataracts Produced by Toxins.—Various toxic agents introduced into the animal body will produce cataract. Thallium, naphthalene, dinitrophenol, dinitro-orthocresol, para-2-chlorobenzene and alloxan, when absorbed into the body, will frequently lead to lens opacities. Such cataracts are rarely described in man but are well known as the result of experimental intoxication in animals.

Two cases of thallium cataracts in human beings are described by Kubesova.[59] Fitzhugh and Buschke [60] studied the cataractogenic properties of compounds related to naphthalene. Beta-tetralol (1, 2, 3, 4-tetrahydro-β-naphthol) fed to rats caused cataract formation in three weeks. This compound, as well as 1, 2, 3, 4-tetrahydro-2-naphthyl propionate and β-naphthylsalicylate, possessed about four times the cataract-producing power of naphthalene. The development of cataract was not retarded

55. Pau, H.: Über Linsenveränderungen bei Achsenmyopia und in Alter, Arch. f. Ophth. **149**:431-436, 1948.

56. Legrand, J.: Punctate Opacities and Tuberculosis, Ann. d'ocul. **183**:58-63, 1950.

57. Auricchio, G.: Studi sulla cataratta complicata: Sulla patogenesi della cataratta complicata secondaria ad iridociclite, Ann. ottal. e clin. ocul. **75**:89-99, 1949.

58. Mann, I.: Recent Cancer Research and Its Relation to Ophthalmic Problems, Am. J. Ophth. **33**:1064-1068, 1950.

59. Kubesova, J.: Neuritis retrobulbaris a katarakta po otrave thalliem, Česk. ofth. **5**:149-153, 1949.

60. Fitzhugh, O. G., and Buschke, W. L.: Production of Cataract in Rats by β-Tetralol and Other Derivatives of Naphthalene, Arch. Ophth. **41**:572-582 (May) 1949.

by diets containing high concentrations of protein, methionine, cystine, riboflavin or ascorbic acid. Pronounced changes in the fluorescence of the rat lens occur after the administration of naphthalene (page 470).

Davids [61] assumed that necrobiosis of certain epithelial cells and fibers of the lens is followed by formation of abnormal cells, which leads to the production of cataracta complicata. He recommends the use of calcium and A T 10 (a proprietary preparation of dihydrotachysterol) to decrease the permeability of the lens epithelium, capsule and fibers. Epinephrine, which diminishes the number of mitoses, may aid in preventing the proliferation of pathological epithelial cells.

Changes Produced by Trauma and Occupation.—In spite of increased knowledge and the employment of preventive measures, occupational diseases and injuries of the eye are still common. Ten cases of cataract have been reported among cyclotron workers (see "radiation cataract"). Minton [62] reports 131 compensation cases of cataract among persons in the metal and chain making industry between 1939 and 1945. Although exposure to the radiation of the welder's arc usually resulted in nothing more serious than conjunctivitis and superficial keratitis, lens opacities similar in type to those caused by heat were occasionally observed, especially in elderly welders.

Rosen [63] describes a case of argyrolentis in a 42 year old dentist, who in his work had been "pushing amalgams" for many years. The alteration consisted of a 3 by 3.5 mm. metallic disklike opacity, which could be resolved into very small grayish blue circles. Faint raylike opacities extended out from the periphery of the disk, producing a sunflower effect. The opacity was confined to the pupillary area.

Radiation Cataract.—It has long been known that exposure to various types of radiant energy may produce cataract. With the reports that cataracts developed in certain persons in Japan who survived exposure to radiations from the atomic blast and in scientists in the United States exposed to cyclotron-induced radiation, the subject of radiation cataract has assumed an importance greater than ever.

Cogan, Martin and Kimura [64] report 10 cases of cataract in Japan induced by radiations from explosions of the atomic bombs. All the persons affected were within 550 to 950 M. of the center of the explosion and were exposed to neutron and gamma rays. The opacities, which seemed to be confined to the posterior capsule, were lacelike in texture, with a few polychromatic crystals and vacuoles. Vision began to fail one-half to 2½ years after exposure.

A report that 10 persons exposed to radiations from the cyclotron manifested varying degrees of posterior cortical opacities in the lens is made by Abelson and Kruger.[65] The patients were divided into three groups. In group 1 were three men with severe changes in the posterior cortex consisting of thick saucer-shaped opaci-

61. Davids, B.: Zur Entstehung des komplizierten grauen Stars (Cataracta complicata) mit Bemerkungen über das Starproblem im allgemeinen und über die konservative Startherapie, Arch. Augenh. **149**:156-174, 1949.

62. Minton, J.: Occupational Eye Diseases and Injuries, J. Roy. Inst. Pub. Health & Hyg. **12**:257-262, 1949.

63. Rosen, E.: Argyrolentis, Am. J. Ophth. **33**:797-800, 1950.

64. Cogan, D. G.; Martin, S. F., and Kimura, S. J.: Atomic Bomb Cataracts, Science **110**:654-655, 1949.

65. Abelson, P. H., and Kruger, P. G.: Cyclotron-Induced Radiation Cataracts, Science **110**:655-657, 1949.

ties. The capsule and nucleus were clear. Group 2 consisted of four men whose visual acuity was only partially reduced, as a result of slight to moderate opacities in the posterior cortex. In group 3 there were three men with minimal changes in the lens, consisting of thin, lacy opacities or punctate dots in the posterior cortex and capsular iridescence.

Reese, Merriam and Martin,[66] reporting on the treatment of retinoblastoma with x-rays, state that the average time for the development of posterior cortical cataract after irradiation was three years two months.

The effect of experimental irradiation of the entire body of newborn mice was the subject of investigations undertaken by Lorenz and Dunn.[67] Using x-rays, they administered a single dose of 400 r and found that lesions developed in all surviving animals in one or both eyes. The most striking changes were atrophy of the retina and lenticular and corneal opacities. This experiment gives further proof of the relatively high degree of sensitivity of young lenticular and retinal tissues to radiant energy.

Daily, Wakim, Herrick, Parkhill and Benedict[68] investigated the effects of microwave diathermy on the intact and enucleated eyes of dogs. Anterior cortical cataracts were produced by a daily exposure of 30 minutes for seven days in one animal and for eight days in another. The authors explain the development of lens opacities without accompanying alterations in the intraocular structures by the probable fact that because the lens and surrounding media are avascular the mechanism for heat loss is relatively less efficient than that in vascular tissues.

Glaucoma and Cataract.—Glaucoma frequently leads to the formation of lens opacities, and, vice versa, certain alterations in the lens may cause or be associated with an increase in the intraocular tension. Thus, cataract is found as a complication of absolute glaucoma, of an acute attack of glaucoma or of glaucoma surgery. Lenticular changes, such as intumescence, dislocation or exfoliation of the capsule, may, on the other hand, give rise to complicating glaucoma.

The goniocycloscopic findings in cases of glaucoma, cataract and other pathological conditions of the eye are reported by von Fieandt.[69] He reports five cataracts in 41 eyes on which cyclodialysis had been performed for simple glaucoma. The opacities appeared 1½ to five years after the operation. Among 12 cases of iridencleisis, there appeared one case of cataract. Since this eye had previously undergone cyclodialysis, however, it could not be established which operation was responsible for the cataract. Von Fieandt also undertook gonioscopic examination before and after operations for senile cataract. The author states that before the operation, except for narrowing of the angle, caused by swelling of the lens, the eyes did not differ from the normal. An important observation was that there were fewer anterior synechias after the extracapsular method with iridotomy than after the

66. Reese, A. B.; Merriam, G. R., and Martin, H. E.: Treatment of Bilateral Retinoblastoma by Irradiation and Surgery, Am. J. Ophth. **32**:175-190, 1949.

67. Lorenz, E., and Dunn, T. B.: Ocular Lesions Induced by Acute Exposure of the Whole Body of Newborn Mice to Roentgen Radiation, Arch. Ophth. **43**:742-749 (April) 1950.

68. Daily, L., Jr.; Wakim, K. G.; Herrick, J. F.; Parkhill, E. M., and Benedict, W. L.: The Effects of Microwave Diathermy on the Eye: An Experimental Study, Am. J. Ophth. **33**:1241-1254, 1950.

69. von Fieandt, O.: Gonio-Cycloscopic Findings in Glaucoma, Cataract and Some Other Pathological Conditions of the Eye, Acta ophth. 1949, supp. 34, pp. 1-132.

intracapsular extraction with iridotomy and that each of these procedures was followed by fewer anterior synechias than was extracapsular or intracapsular extraction with iridectomy. Therefore, an additional advantage of preservation of the round pupil is the decreased likelihood of secondary glaucoma produced by anterior synechias obstructing the chamber angle.

Cataracta Disseminata Subepithelialis Glaucomatosa Acuta.—Vogt was the first to describe accurately the appearance of lens opacities which immediately follow an acute attack of glaucoma. Nemetz [70] reports 102 cases in which lens opacities were observed after an acute attack of glaucoma. The size, as well as the number of some of the opacities, which, incidentally, were not related to the severity of the glaucomatous attack, decreased with time, indicating that the lesions were reversible. Three to six months later the opacities were seen deeper in the cortex and were of a permanent nature. Nemetz disagrees with Sugar's earlier statement that the typical opacities are found only in the pupillary area, for in 12 of his cases the opacities were observed in the colobomatous area. The author assumes that these opacities, as well as similar opacities which have been reported to follow blast explosion, ammonia injury, cauterization with 20 per cent zinc sulfate and penetrating injuries, are caused by the release of histamine or histamine-like substances.

Glaucoma Capsulare.—After examining 57 eyes with simple glaucoma, Thomassen [71] reports that 45, or 79 per cent, showed evidence of exfoliation of the lens capsule.[72] Sugar [73] describes a group of cases of secondary glaucoma caused by spontaneous rupture of the lens capsule. The increased intraocular tension which followed the rupture was attributed to the chemical irritation of the escaping lens substance.

CATARACT SURGERY

The literature for 1949-1950 dealing with the various phases of cataract surgery is so extensive that only the most important papers are included in this review.

The postgraduate student, the hospital resident and the established surgeon will find Kirby's [74] "Surgery of Cataract" a valuable book. This well illustrated volume gives a comprehensive account of the development of cataract surgery and the preoperative preparations. The author highlights the development of the technique of the present day intracapsular extraction and then proceeds to describe his own methods of intracapsular cataract extraction.

Callahan,[75] in "Surgery of the Eye: Injuries," includes an excellent section on the surgical treatment of traumatic cataract and dislocation of the lens. The surgical technique is described in an interesting manner, and the major procedures are splendidly illustrated.

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70. Nemetz, U.: Zur Frage der Cataracta glaucomatosa acuta, Klin. Monatsbl. Augenh. **115**:417-421, 1949.

71. Thomassen, T. L.: On the So-Called Capsular Glaucoma, Acta ophth. **27**:423-427, 1949.

72. Authorities vary widely in their findings: 9 per cent by Vogt, 13 per cent by Gradle and Sugar and up to 86 per cent by Horren.

73. Sugar, H. S.: Acute Secondary Glaucoma Due to Spontaneous Rupture of the Lens Capsule, Am. J. Ophth. **32**:1509-1513, 1949.

74. Kirby, D. B.: Surgery of Cataract, Philadelphia, J. B. Lippincott Company, 1950.

75. Callahan, A.: Surgery of the Eye: Injuries, Springfield, Ill., Charles C Thomas, Publisher, 1950.

Preparation of the Patient.—Post [76] discusses the measures necessary to prevent infection in eye surgery and emphasizes the importance of the proper care and sterilization of the instruments. The need for the proper preparation of the whole patient for cataract surgery is emphasized by Kirby.[77] In the case of one-eyed cataractous patients Kirby [78] urges that extra care and precautions be taken in the preoperative, operative and postoperative stages. Zaushkina [79] shows that the prophylactic use of vitamins A and K (ascorbic acid and menadione) did not decrease the frequency of postoperative hemorrhages into the anterior chamber.

Anesthesia.—Parrish and associates [80] describe the use of general anesthesia for ophthalmic surgery. The authors believe that the use of an endotracheal catheter is the only procedure which will assure a patent airway. This permits control of the anesthesia even if the anesthesiologist is removed from the surgical field. Sanders and Cutler,[81] who employs a general anesthesia consisting of an intravenous injection of thiopental (pentothal®) sodium combined with the inhalation of 65 per cent nitrous oxide, 30 per cent oxygen and 5 per cent cyclopropane, discuss the advantages and disadvantages of this form of general anesthesia. The advantages mentioned are the decreased preoperative apprehension; elimination of the possibility of retrobulbar hemorrhage resulting from retrobulbar injection; avoidance of lid squeezing, caused by incomplete seventh nerve block; elimination of ocular movements, and, finally, a moderate reduction in intraocular tension. The disadvantages appear to be chiefly the increased time consumed in preparing the patient for operation and the greater incidence of hyphemia, postoperative nausea and vomiting.

Reports on the use of curare in cataract surgery have appeared (Kirby,[82] Roche [83] and others). The use of curare in combination with local anesthesia and with thiopental sodium is described by Kirby. Because of the dangers inherent in the use of curare, the reviewer agrees with Cordes [84] that curare is not to be used in the usual case of cataract extraction.

Atkinson [85] states that the addition of hyaluronidase to procaine hydrochloride increased the rapidity of local anesthesia and the onset of akinesia, induced either

76. Post, M. H.: Prevention of Infection in Eye Surgery, Am. J. Ophth. **32**:679-685, 1949.

77. Kirby, D. B.: The Preparation of the Whole Patient for Cataract Surgery, Irish J. Med. Sc. 1949, pp. 707-714.

78. Kirby, D. B.: Cataract Surgery for One-Eyed Patients, Tr. Am. Acad. Ophth., 1950, pp. 542-560.

79. Zaushkina, N.: The Influence of Vitamins C and K on the Frequency of Hemorrhages After Cataract Extraction, Vestnik oftal. **27**:30-31, 1948.

80. Parrish, R. G.; Esson, E., and Karp, M.: General Anesthesia for Ophthalmic Surgery, Am. J. Ophth. **33**:883-889, 1950.

81. Sanders, R. D., and Cutler, N. L.: General Anesthesia in Cataract Surgery, Arch. Ophth. **43**:653-660 (April) 1950.

82. Kirby, D. B.: Use of Curare in Cataract Surgery, Arch. Ophth. **43**:678-693 (April) 1950.

83. Roche, J. R.: Research in the Use of Curare for Ocular Surgery, Am. J. Ophth. **33**:91-97, 1950.

84. Cordes, F. C., in discussion on Kirby,[82] pp. 691-693.

85. Atkinson, W. S.: Use of Hyaluronidase with Local Anesthesia in Ophthalmology: Preliminary Report, Arch. Ophth. **42**:628-632 (Nov.) 1949.

by the Van Lint or by the O'Brien procedure. Lebensohn [86] stresses the fact that marked hypotony is produced by the addition of hyaluronidase to the usual solution employed for retrobulbar anesthesia. He states that this combination is contraindicated in the uncomplicated type of cataract because the normal vitreous pressure plays an important role in the mechanics of the intracapsular extraction.

Operative Techniques.—That there are still considerable variations in the actual operative procedures is evident from a perusal of the literature. The great majority of the recent writers, however, agree on the advantage of the intracapsular operation over that of the extracapsular. The intracapsular extraction with forceps and pressure is performed either by grasping the capsule superiorly and sliding the lens out or by grasping the capsule inferiorly and tumbling the lens. Kirby,[87] Vannas [88] and Anthony [89] are among the ophthalmologists who have described their method of removing the lens by grasping the capsule from above. The chief advantage of this method is that it makes it possible for the surgeon to have a direct view of the lens as it is being dislodged. Anthony employs the Verhoeff extraction, preceded by a complete iridectomy before grasping the lens. Kirby and Vannas, who prefer round pupil extractions, describe their methods of overcoming the difficulties encountered in seizing the lens capsule from above. Kirby prefers to apply the forceps tangentially at the upper portion of the lens capsule just anterior to the equator, because this allows for more natural movements of the hand and arm. When the zonule is very resistant to ordinary indirect pressure, traction and rotation, Kirby recommends that direct separation of the zonule from the capsular attachments be done. Vannas produces eccentric mydriasis first by rubbing glaucosan (a preparation of epinephrine and histamine) on the surface near the limbus and, later, by direct swabbing along the lens capsule and iris. This procedure permits him to apply the forceps to the lens capsule near the upper equator. Both authors stress the importance of applying pressure with the tip of an expresser rather than with the broad surface, since the latter increases the intraocular tension out of proportion to the increased stripping effect on the zonular fibers. Vannas employs a triangular suture, the corneal part of which resembles the Liégard suture and the sides of which are formed by inserting the needle and thread tangentially through the conjunctiva and superficial layer of the sclera toward the vertical meridian.

Davis,[90] in an excellent presentation, compares the final results obtained from knife and keratome incisions. The number of complications were less and healing was more rapid when the incisions were made with the knife than when they were made with the keratome. The author emphasizes the importance of deep sutures when the round pupil extraction was performed with either type of incision.

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86. Lebensohn, J. E.: Hyaluronidase in Ocular Surgery and Therapy, Am. J. Ophth. **33**:865-870, 1950.

87. Kirby, D. B.: The Rupture of the Zonule in Intracapsular Cataract Extraction: A New Method, Brit. J. Ophth. **33**:3-21, 1949; Direct Separation of the Zonule, Tr. Ophth. Soc. U. Kingdom **49**:595-606, 1949; The Various Methods of Separation of the Zonule for Intracapsular Cataract Extraction, Arq. port. oft. **2**:3-31, 1950.

88. Vannas, M.: The Intracapsular Cataract Operation—Experiences and Suggestions, Ophthalmologica **118**:866-878, 1949.

89. Anthony, A. R.: An Analysis of 793 Cataract Operations by the Verhoeff Intracapsular Sliding Method, J. Internat. Coll. Surgeons **12**:556-568, 1949.

90. Davis, F. A.: Incision and Closure of the Wound in Cataract Operations: A Study of the Graefe Knife Versus the Keratome-Scissors Incision and a Method of Suture of the Deep Wound, Arch. Ophth. **44**:175-197 (Aug.) 1950.

Wolfe and associates [91] describe their method of corneal incision in 300 consecutive cases of cataract extraction. They state that their technique, consisting of an incision in the clear cornea with five preplaced sutures, has the advantage that it reduces the incidence or severity of hyphema, astigmatism and iris prolapse and that, if secondary glaucoma should develop, there is a better possibility for a successful filtering operation.

Barraquer [92] relates his experiences in more than 20,000 cataract extractions. He makes use of a special keratome, an iridectomy at 11 and 2 o'clock, conjunctivasclerocorneal sutures and the eresiphake. Moreno [93] and Pérez Llorca [94] also report on their use of the suction method for cataract removal. Muir and Kafka [95] describe their method of exposure of the globe in cataract surgery in order to avoid the dangers inherent in use of the ordinary speculum. Their technique requires the use of a Katzin aluminum suture mask and modified blades of the Guyton-Parks speculum. A new method for retraction of the upper lid, by passing two sutures through the entire thickness of the upper eyelid just above the tarsal plate and then fastening them to form a sling or a hammock, is recommended by Quinn and Stansbury.[96]

In an excellent article, Harrington [97] discusses the results of his investigations of the mechanics of intracapsular cataract extraction. He reports that the rupture of the zonules is brought about chiefly by the wedge of vitreous which is made to insert itself between the ciliary processes and the lens equator. The vitreous acts as a tissue, and not as a fluid, which would transmit pressure equally in all directions. He shows that rupture of the zonules does not ordinarily occur from traction alone.

Closure of Incision for Cataract Operation.—Corneal sections may be closed by means of physiological glue or sutures or by a combination of the two methods. Tassman,[98] reporting on his experimental studies, states that the use of coagulum eliminates the postoperative use of sutures because the wound seals immediately and the anterior chamber reforms in most instances within 30 minutes. This reduces the danger of postoperative prolapse of the iris, and recovery is safer and more rapid. The reports by Town and Naidoff [99] are in essential agreement with

91. Wolfe, O. D.; Wolfe, R. M., and Wolfe, O. R.: Corneal Incision for Cataract: Author's Technique, J. Internat. Coll. Surgeons **13**:415-428, 1950.

92. Barraquer, I.: Experiencia de mas de veinte mil operaciones de cataracta, Clín. laborat. **47**:401-409, 1949.

93. Moreno, J.: La facoerisis simplificada, Arch. Soc. oftal. hispano-am. **9**:508-518, 1949.

94. Pérez Llorca, J.: Sobre la operación de la catarata, Arch. Soc. oftal. hispano-am. **9**:501-507, 1949.

95. Muir, B. W., and Kafka, A. J.: A New Method for Exposure of the Globe in Cataract Surgery, Am. J. Ophth. **33**:1146-1148, 1950.

96. Quinn, L. H., and Stansbury, J. R.: A Sling Retraction Suture for the Upper Lid During Cataract Surgery, Am. J. Ophth. **33**:441-447, 1950.

97. Harrington, D. O.: Mechanics of Intracapsular Cataract Extraction, Arch. Ophth. **42**:23-41 (July) 1949.

98. Tassman, I. S.: Experimental Studies with Physiologic Glue (Autogenous Plasma plus Thrombin) for Use in the Eyes, Am. J. Ophth. **33**:870-818, 1950.

99. Town, A. E., and Naidoff, D.: Fibrin Closure in Eye Surgery, Am. J. Ophth. **33**:879-882, 1950. Town, A. E.: Fibrin Closure in Surgery of the Eye, J. Internat. Coll. Surgeons **14**:114-118, 1950.

Tassman's conclusions. Von Grósz [100] found three instances of wound rupture following the use of thrombin alone for closure of corneal wounds.

Bell [101] reviews the types of sutures employed in cataract surgery and classifies them as follows: the conjunctivoconjunctival, the corneoconjunctival, the sclero-conjunctival and the corneoscleral-conjunctival. Three methods of corneoscleral suturing are available, according to Laval.[102] These are as follows: (1) preplaced sutures, (2) sutures placed after the section and (3) sutures placed partly before and partly after the section. The author then proposes a new type which is a compromise between the suture that is preplaced and the suture that is placed after the section is made. The compromise consists in placing the suture after performing a 5 mm. keratome section. Papolczy [103] employs a 000000 black nylon suture. He inserts the suture 1 mm. from the limbus at 12 o'clock. Then the cataract incision is made with a von Graefe knife up to the level of the suture, at which point the knife is withdrawn. This is followed by the preparation of a semicircular conjunctival flap, which is then detached from the sclera and turned back on the cornea. The threads are drawn out through the conjunctiva, and a loop is formed. When the lens is removed, the flap is replaced, and the threads are tied. Preparatory wound sutures result in a less degree of postoperative astigmatism and a decreased number of wound ruptures than do other types of wound closures.[104]

Immediate Postoperative Care.—Pilocarpine, instead of physostigmine, is the agent of choice when a miotic is necessary after cataract extraction. Scheie and Ojers [105] showed experimentally that physostigmine is not effective after retrobulbar injection, while pilocarpine produces a prompt miosis. Since pilocarpine is not effective for several hours if homatropine has been used prior to operation, the authors recommend that hydroxyamphetamine hydrobromide (paredrine® hydrobromide) be employed as the preoperative mydriatic. Gerber [106] advocates that 10,000 units of penicillin be injected subconjunctivally after cataract operation in order to prevent postoperative infections.

Congenital and Secondary Cataract.—According to Bagley,[107] in congenital cataract the results of needlings are definitely inferior to those following a combination of needling and lavage or linear extraction. Because of the early and late complications resulting from discission followed by extraction of congenital cataract,

100. von Grósz, S. Z.: Biologisches Verfahren zur Sieherung der Wunde bei Staroperationen, Klin. Monatsbl. Augenh. **115**:393-395, 1949.

101. Bell, D. P.: Sutures Used in Cataract Surgery: A Review, Am. J. Ophth. **32**:639-649, 1949.

102. Laval, J.: A Simple and Efficient Cataract Suture, Arch. Ophth. **42**:189-191 (Aug.) 1949.

103. Papolczy, F.: Statistical Data of My Cataract Operation Performed with a New Suture of the Sclera, Brit. J. Ophth. **33**:296-305, 1949.

104. Harmuth, E.: Über Einfluss des Wundverschlusses auf den Endastigmatismus und Wundheilung nach Starausziehung, Wien. klin. Wchnschr. **61**:606, 1949.

105. Scheie, H. G., and Ojers, G.: Choice of a Miotic Agent Following Retrobulbar Anesthesia, Am. J. Ophth. **32**:1369-1375, 1949.

106. Gerber, H.: Penicillin prophylaxe bei der Kataraktoperation, Wien. klin. Wchnschr. **61**:685-686, 1949.

107. Bagley, C. H.: Congenital Cataracts—A Survey of the Various Types of Operation, Am. J. Ophth. **32**:411-419, 1949.

Denig [108] advocates that the first procedure be an optical iridectomy and, if this is not adequate, a loop extraction be performed eight weeks later.

Raiford [109] compared experimental incisions on rabbits as follows: (1) incision through the lens capsule only, (2) discission extending through the cortex and nucleus and (3) discission through the entire lens and into the vitreous. As might have been anticipated, the second type of discission was most satisfactory and gave the minimum side reactions. In the case of secondary membranous cataract, Fontana [110] recommends the von Graefe discission, except that when there is a dense membrane the membrane may be extracted by means of forceps. However, when there is a dense iridocapsular membrane, a punch forceps is preferable.

Cataract Secondary to Uveitis and Trauma.—Meisner [111] reports the results of cataract extraction in 32 patients who had had chronic uveitis. There were 25 women most of whom were of advanced age and nearly all of whom had the disease in both eyes. The condition was unilateral in 4 of the seven men. The author recommends that the operation not be performed until long after all signs of inflammation have subsided. Because the zonules were usually atrophic, the intracapsular operation was easily performed in the majority of cases and was surprisingly free from complications. The poorest results were obtained in eyes complicated by glaucoma, retinal detachment, choroiditis or vitreous opacities.

Pillat [112] reports 105 cases of intracapsular extraction performed for complicated cataract. Among the causes were iridocyclitis (63 cases), glaucoma (30 cases), heterochromia (5 cases) and aplasia of the iris (2 cases). All the patients tolerated operation exceedingly well, and the number of complications was no greater than in operations for ordinary senile cataract.

The course and results of surgical measures for traumatic cataract in 120 patients during World War II are reported by Barbel.[113] The only new procedure employed was the preparation of a conjunctival flap by making the keratome incision through the conjunctiva at a point 5 to 6 mm. above the limbus. Many of the eyes had other serious injuries, such as intraocular foreign bodies. Despite the severe injuries, the results were considered good in 58 patients. The author concluded that operation should be performed early for traumatic cataract and that the conjunctival flap is a good preventive measure against postoperative infection.

Glaucoma and Cataract.—Lee and Weih,[114] in an important article, analyze the operative procedures in 100 cases of cataract and glaucoma. They found that the

108. Denig, R.: Früh- und Spätfolgen der Diszission-Extraktion des angeborenen Kernstars und seiner Abarten: Rückkehr zur optischen Iridektomie evtl. Schlingenentbindung, Klin. Monatsbl. Augenh. **114**:197-202, 1949.

109. Raiford, M. B.: Cataract Discission Operations: An Experimental Evaluation, Am. J. Ophth. **33**:1055-1063, 1950.

110. Fontana, F.: Il problema operatorio della cataratta secondaria, Rassegna ital. d'ottal. **18**:237-249, 1949.

111. Meisner, W.: Über Cataracta complicata nach chronischer Iridozyklitis, ihre Operation und Prognose, Klin. Monatsbl. Augenh. **115**:543-548, 1949.

112. Pillat, A.: Intracapsular Extraction of Cataracta Complicata Accreta After Iridocyclitis and Glaucoma, Arch. Ophth. **42**:567-582 (Nov.) 1949.

113. Barbel, I. E.: Certain Peculiarities of the Course and Outcome of Operations for Traumatic Cataract, Vestnik oftal. **28**:11-14, 1949.

114. Lee, O. S., and Weih, J. E.: Results of Operation for Cataract with Primary Glaucoma, Arch. Ophth. **44**:275-284 (Aug.) 1950.

intracapsular technique is the operation of choice. When the glaucoma was controlled by miotics, a combined cataract extraction was frequently the only surgical procedure necessary. However, when the tension was not controlled by miotics, an anterior sclerectomy or iridencleisis combined with cataract extraction was the recommended procedure. Sternberg and Meyer [115] believe that increased intraocular tension secondary to intumescence of the lens occurs only in the preglaucomatous, narrow angle type of eye and should be treated by means of an anterior fistulizing operation before the lens is extracted. This opinion is strikingly divergent from the widely accepted belief that removal of the intumescent lens usually leads to complete recovery from the increased intraocular tension.

Gasteiger [116] reports that cataract developed in about 10 per cent of 430 cases of trephination and in about the same percentage of 515 cases of cyclodialysis. Protracted hypotony was not a factor, but excessive massage of the cornea, reposition of the iris by instruments and postoperative iritis favored cataract formation. The author preferred to make an incision immediately anterior to the bleb. Neither a conjunctival flap nor sutures were used. The operation of choice was the intracapsular technique. In general, the tension was unchanged after the operation.

COMPLICATIONS OF CATARACT SURGERY

With the improvement of operative technique, the application of chemotherapeutic and antibiotic agents and the broader understanding of the ophthalmologist, the incidence of complications following cataract surgery has become progressively lower. However, postoperative complications are still too common, and it should be the constant aim and effort of the ophthalmologist to reduce them to the minimum.

A noteworthy article showing the important part that loss of vitreous plays in the production of postoperative complications is that of Knighton.[117] He studied the records of 1,001 consecutive cases of intracapsular extraction and found that in 91 cases the vitreous was lost or presented. Of eyes without vitreous loss, 71 per cent healed normally, while only 21.8 per cent of the eyes with vitreous loss healed uneventfully. Postoperative complications, such as iridocyclitis, iris prolapse, vitreous opacities, glaucoma, keratitis and retinal detachment, were about four times as frequent in eyes which had lost vitreous as in those which had not. The important subject of herniation of the anterior hyaloid membrane following uncomplicated intracapsular cataract extraction is discussed by Reese.[118] The author believes that the herniation is produced by thinning of the anterior hyaloid membrane and an increase in the volume of the vitreous. This condition frequently produces a central disk-shaped opacity of the cornea. Examination with

115. Sternberg, P., and Meyer, S. J.: Choice of Operation in Acute Glaucoma Secondary to Swelling of the Lens, Am. J. Ophth. **33**:763-768, 1950.

116. Gasteiger, H.: Über Kataraktextratraktionen in Augen mit vorher ausgeführter: Elliotscher Trepanation, Klin. Monatsbl. Augenh. **114**:370-385, 1949.

117. Knighton, W. S.: The Role of Vitreous in Postoperative Complications of Intracapsular Extractions, Tr. Am. Ophth. Soc. **47**:606-620, 1949.

118. Reese, A. B.: Herniation of the Anterior Hyaloid Membrane Following Uncomplicated Intracapsular Extraction, Am. J. Ophth. **32**:933-946, 1949.

magnification shows that the hyaloid membrane is in apposition to the cornea, causing irritation. According to Reese, the striate keratitis which follows intracapsular extraction results from this form of herniation. In most instances, especially after round pupil extractions, the herniation will respond to the administration of a strong miotic. If the herniation is very large, it is recommended that the patient lie flat on his back and the pupil be dilated with 10 per cent phenylephrine hydrochloride (neo-synephrine® hydrochloride) and then constricted with a miotic. The injection of air into the anterior chamber is sometimes beneficial.

Velhagen [119] classifies prolapse of the vitreous into the anterior chamber into two types, in one of which the hyaloid membrane is intact and in the other the membrane is ruptured. The consequences of vitreous prolapse are increased intraocular tension, opacification of the cornea and decreased visual acuity. Treatment for increased tension consists of the application of drugs; if this fails, surgical intervention becomes necessary. Injection of air is recommended to separate the prolapsed vitreous from the cornea and thus to prevent the formation of corneal opacities.

In a valuable article Calhoun [120] discusses the diagnosis and treatment of epithelial invasion of the anterior chamber. The author stresses the need for early recognition of the condition if the eye is to be saved. A thin wavy line on the endothelial surface of the cornea, which represents the advancing border of the epithelial downgrowth, is characteristic of this condition. If unrecognized or if treatment is initiated too late, the prognosis is poor. The treatment consists of use of x-radiation, of high voltage and in fractional doses. Pincus [121] reports five cases of epithelization of the anterior chamber. Radiation therapy resulted in improvement in three cases. The author emphasizes the importance of the preformed conjunctival flap in reducing the possibility of implanting epithelium.

In a comprehensive article dealing with the complications of cataract surgery, Clark [122] discusses all the important complications except sympathetic ophthalmia. His report, covering 2,086 cataract extractions, classifies the complications as follows: accidents at operation, postoperative complications and complications resulting from the general state of the patient. The author stresses that the best treatment is prevention.

Delayed reformation of the anterior chamber is considered by Meyer and Sternberg [123] to be the most important factor in the production of secondary glau-

119. Velhagen, K.: Die klinische Bedeutung des Glaskörpervorfalls in die Vorderkammer, Ophthalmologica **118**:173-181, 1949.

120. Calhoun, F. P., Jr.: The Clinical Recognition and Treatment of Epithelization of the Anterior Chamber Following Cataract Extraction, Tr. Am. Ophth. Soc. **47**:498-533, 1949.

121. Pincus, M. H.: Epithelial Invasion of Anterior Chamber Following Cataract Extraction: Effect of Radiation Therapy, Arch. Ophth. **43**:509-519 (March) 1950.

122. Clark, W. B.: The Important Complications of Cataract Surgery (Exclusive of Sympathetic Ophthalmia), New Orleans M. & S. J. **102**:248-259, 1949.

123. Meyer, S. J., and Sternberg, P.: Secondary Glaucoma After Cataract Extraction, Tr. Am. Acad. Ophth. 1950, pp. 326-339.

coma when conjunctival sutures are used. However, when corneoscleral sutures were employed, iridocyclitis was a factor equal in importance to delayed reformation.

The most effective treatment of postoperative infections according to Dellaporta and his associates [124] is the combination of injections of milk intramuscularly and of penicillin subconjunctivally. Four patients treated in this manner recovered satisfactorily.

Vrany [125] compares the results in 162 cases of cataract extraction in which corneoscleral sutures were not used but in which canthotomy was performed. He states that the incidence of iris prolapse in his series was 1.8 per cent, a percentage comparable to that found when corneoscleral sutures were used. He believes that canthotomy plays an important part in the prevention of iris prolapse. Gürtler [126] reports that in 27 cases of intraocular operations, most of which were cataract extractions, cotton wool threads were found in the anterior chamber. These produced very little irritation or secondary reaction.

A complication that is seldom mentioned is the dislodgment of flakes of pigment 1 mm. or more in diameter during intracapsular operation. These flakes may seriously impair the visual acuity. Atkinson [127] recommends that these pigment flakes be removed at the time of the operation whenever possible.

Dunnington and Regan [128] discuss the diagnosis and treatment of late fistulization, in contrast to the usual type, which is large and open at all times. The diagnosis of a slow or intermittent leakage is made on the basis of lacrimation, blurred vision and hypotony. If gentle but firm pressure is made on the eye after the instillation of fluorescein, leakage of aqueous becomes visible. The treatment recommended by these authors consists of a combination of cauterization of the opening, followed by suturing the wound and covering it with a conjunctival flap.

VITREOUS HUMOR

Several valuable contributions have appeared during the past year dealing with the vitreous humor.

Hyaluronic Acid and Hyaluronidase.—One of the important subjects investigated during the past year was the role of hyaluronic acid in the physiology and pathology of the eye. Hyaluronic acid is a mucopolysaccharide, which is capable of binding water in the interstitial spaces in animal tissue. It is an important component of the vitreous body. The enzyme which hydrolyzes hyaluronic acid is known as hyaluronidase. In the eye this enzyme is found in the ciliary body and iris. Pirie [129] considers the vitreous to be composed of a network of fibrils

124. Dellaporta, A.; Riemer, F., and Harmuth, E.: Eine vergleichende Untersuchung über die Infektionshaüfigkeit nach Kataraktoperation in den Jahren 1936, 1941, 1946 and 1948, mit Berücksichtigung der Penicillinbehandlung, Klin. Monatsbl. Augenh. **115**:369-384, 1949.

125. Vrany, O.: Prehled komplikací po operâcich katarakt na Plzenské öcní klinice za poslendí tri roky, Česk. ofth. **5**:164-168, 1949.

126. Gürtler, E.: Watterfäden in der Vorderkammer nach bulbuseröffnenden Operationen, Wien. klin. Wchnschr. **61**:686-689, 1949.

127. Atkinson, W. S.: Flakes of Iris Pigment on Posterior Surface of Cornea Following Cataract Operation, Arch. Ophth. **43**:32-35 (Jan.) 1950.

128. Dunnington, J. H., and Regan, E. F.: Late Fistulization of Operative Wounds: Diagnosis and Treatment, Arch. Ophth. **43**:407-418 (March) 1950.

129. Pirie, A.: Ox Vitreous Humor: Hyaluronic Acid Relationship, Brit. J. Ophth. **33**:271-273, 1949.

and a viscous fluid of hyaluronic acid. Experiments conducted by Pirie indicate that the soluble proteins of the vitreous wash out more readily than does the hyaluronic acid. However, since the latter does not separate entirely from the protein, it is possible that a hyaluronic acid and protein compound may exist in the vitreous. Pirie shows how these two components protect each other: Aggregated hyaluronic acid retards liquefaction of the insoluble protein, and the latter retards the disaggregation of the former. In a second series of experiments, Pirie [130] confirms an earlier report by von Sallmann that hyaluronidase injected into the vitreous humor of the rabbit caused an inflammatory reaction. Pirie found that after such an injection of hyaluronidase the hyaluronic acid of the vitreous became rapidly disaggregated and remained in a nonviscous form for about one month. After this time the hyaluronic acid of the vitreous regained its normal aggregated condition. These observations indicated that there is a continuous production of hyaluronic acid in the living eye. The author also found that after the injection of hyaluronidase in the vitreous of the rabbit the intraocular tension is reduced for several days and that there is an increased concentration of nitrogen in the vitreous and aqueous humors. In a series of important experiments, von Sallmann [131] showed that the injection into the vitreous of hyaluronidase prepared from bull testis accelerated the disappearance of red blood cells but produced only transitory iritis, without signs of any notable infiltration of the posterior segment. The increased rate of red blood cell absorption produced by hyaluronidase is apparently due to increased phagocytosis. Since similar results were produced with heat-inactivated enzyme, the investigator believes that a nonspecific, heat-resistant factor is present in the preparation of hyaluronidase which has the properties of causing an inflammatory response in the eye and accelerating the destruction and transportation of red blood cells and their products.

Godtfredsen [132] examined the subretinal fluid in cases of primary, severe retinal detachment and found that it contains a considerable concentration of hyaluronic acid. In contrast, he found that the subretinal fluid in cases of malignant melanoma of the choroid with secondary detachment was not viscid and hyaluronic acid was absent. Thus, the absence or presence of hyaluronic acid in the aspirated subretinal fluid may serve as an aid in the differential diagnosis of primary retinal detachment and the detachment secondary to a neoplasm. The large amount of hyaluronic acid in retinal rupture with detachment favors the hypothesis that fluid is derived from the vitreous. On the basis of his findings, the author offers a new hypothesis for the pathogenesis of retinal detachment, namely, that local ischemia is followed by tissue necrosis, cystoid degeneration and retinal rupture. Hyaluronidase liberated in the ischemic area produces depolymerization of the hyaluronic acid in the

130. Pirie, A.: The Effect of Hyaluronidase Injection on the Vitreous Humour of the Rabbit, Brit. J. Ophth. **33**:678-684, 1949.

131. von Sallmann, L.: Experimental Studies on the Vitreous: I. Experiments on Diffusion in the Vitreous and on Permeability of Its Surface Condensation Layer, Arch. Ophth. **42**:583-595 (Nov.) 1949.

132. Godtfredsen, E.: Investigation into the Hyaluronic Acid and Hyaluronidase in the Subretinal Fluid in Retinal Detachment, Partly Due to Ruptures and Partly Secondary to Malignant Choroidal Melanoma, Brit. J. Ophth. **33**:721-731, 1949.

adjacent vitreous humor, making the latter less viscid and permitting this substance to penetrate subretinally. Then, when a balance between the two portions of vitreous humor is reached, the depolymerization of the hyaluronic acid ceases.

Phosphate.—The phosphate content of vitreous humor was determined by Palm [133] and found to be equal to about one-third that of aqueous humor. Earlier investigators had reported equal values for phosphate in the aqueous humor and the vitreous body. The phosphate content of the anterior vitreous humor adjacent to the aqueous humor and ciliary body approaches that of the aqueous humor. According to Palm, these findings indicate that the fluid permeating the vitreous body is not identical with the aqueous humor. Since it has been shown that the glucose content of the vitreous is also less than that of the aqueous, due to the consumption of the glucose by the retina (Adler), the author postulates that the glucose and the phosphate leave the vitreous humor by the process known as phosphorylation.

Penetration into the Vitreous.—Davson and associates [134] studied the relative rates of penetration into the vitreous of sodium, potassium, thiocyanate, phosphate, amino acetic acid, alanine, creatinine and urea. They found that these substances reach the vitreous from the ciliary body, the choroid, the pars optica retinae, the pigmented epithelium and the retinal circulation. Potassium, sodium, amino acids and creatinine enter chiefly through the ciliary body. Thiocyanate, which is an ion at the extreme end of the lyotropic series, permeates readily biologic membranes and penetrates into the vitreous body from both the ciliary body and the choroid. Monosaccarides and urea, like thiocyanate, also enter the vitreous from the ciliary body and the posterior region. These experiments show that the chemical structure of a substance is very important in determining its rate of penetration.

Von Sallmann and associates [135] studied the eye with radiosodium and radioiodine autographs. Radiosodium appeared in the region of the ciliary body, choroid and optic nerve. The main entrance of this substance into the vitreous appears to be from the ciliary body and from the region anterior to the optic nerve. Radiosodium injected intravitreally diffused through more than two thirds of the vitreous body within one hour and was uniformly distributed through the vitreous in about four hours. Later the region of the ciliary body and optic nerve showed an accumulation of this substance, indicating a preferential route of absorption. The rate of diffusion was lower in the enucleated eye. This evidence supports the theory that there is a through and through circulation in the vitreous. Radiosodium applied by means of ion transfer apparatus diffused into the anterior chamber but

133. Palm, E.: The Phosphate Content of the Vitreous Body, Acta ophth. **27**:553-562, 1949.

134. Davson, H.; Duke-Elder, W. S.; Maurice, D. M.; Ross, E. J., and Woodin, A. M.: The Penetration of Some Electrolytes and Non-Electrolytes into the Aqueous Humour and Vitreous Body, J. Physiol. **108**:203-217, 1949. Duke-Elder, S.; Davson, H., and Maurice, D. M.: Studies on the Intraocular Fluids: II. The Penetration of Certain Ions into the Aqueous Humor and Vitreous Body, Brit. J. Ophth. **33**:329-338, 1949.

135. von Sallmann, L.; Evans, T. C., and Dillon, B.: Studies of the Eye with Radiosodium Autographs, Arch. Ophth. **41**:611-626 (May) 1949. von Sallmann, L., and Dillon, B.: Studies of the Eye with Radioiodine Autographs, Am. J. Ophth. **33**:429-440, 1950.

into the vitreous only in aphakic eyes. Radioiodine introduced intraperitoneally localizes preferentially in the cornea. However, it is found in the vitreous humor, as well as in other ocular tissues. This amount is slightly increased in eyes with intravitreal infections. In other experiments, von Sallmann investigated the diffusion into the vitreous body of hemoglobin in weak and strong acids and bases. He found that the diffusion of homologous hemoglobin was accelerated by temperature increments to a greater extent in the (less viscid) pig vitreous than in steer vitreous. Decrease of viscosity in autolyzed preparations caused more rapid diffusion of hemoglobin. Hyaluronidase added to hemoglobin or to Niagara blue did not increase the rate of diffusion in the vitreous. Variations in the hydrogen ion concentration of vitreous within the range of pH 6 and 8 did not alter the rate of diffusion of hemoglobin.

Slit Lamp Examination of Vitreous.—Goldmann [136] describes his method of examining the vitreous and fundus by means of the slit lamp and a contact glass made of plastic. He found that pathological alterations can be discovered earlier by this means than by the usual methods. In vitreous detachment he decribes the vitreous framework as being condensed inferiorly, with an optically empty space lying above. In cases of retinal detachment biomicroscopy will often show the mechanism of the origin of the detachment. In inflammations of the posterior region, a Tyndall phenomenon is observable in the posterior vitreous space. In periphlebitis retinae and at the onset of other retinal diseases, such as chorioretinitis, he observed that the posterior limiting membrane of the vitreous was detached from the retina and became visible as a fine grayish line.

Bottoni and Raimondo [137] studied the behavior of the rabbit eye after transplantation of vitreous. Withdrawal of 0.5 to 1.1 ml. of vitreous is tolerated by a 2 Kg. rabbit. Gradually the intraocular tension returns to normal. Fresh homologous vitreous may be safely substituted in amounts ranging from 0.5 to 0.8 ml. The vitreous was observed to be clear within two weeks.

Pooled human vitreous which has been refrigerated at temperatures between 32 and 50 F. was found by Fritz [138] to produce little reaction and no interference with the visual acuity in the recipient's eye. Fritz suggests that this method would simplify the Cutler procedure of vitreous transplantation, which requires that the recipient and the donor be operated on simultaneously. Other than the replacement of pathological vitreous, the main indication for vitreous substitution is the hypotony which may follow retinal detachment, cataract extraction or injury.

The difficulty in achieving an adequate concentration of antibiotic in the vitreous by means other than direct intravitreal injection is well known. In fact, an antibiotic to which an organism is only partially susceptible, if injected intravitreally, may be more effective in overcoming infections of the vitreous than an antibiotic superior from the point of view of the organism's sensitivity when

136. Goldmann, H.: Slitlamp Examination of the Vitreous and the Fundus, Brit. J. Ophth. **33**:242-245, 1949.

137. Bottoni, A., and Raimondo, N.: Ricerche sperimentali sul trapianto di vitreo, Ann. ottal. e clin. ocul. **75**:311-319, 1949.

138. Fritz, M. H.: The Use of Sterile Refrigerated Pooled Human Vitreous in Living Eyes, Am. J. Ophth. **32**:45-50, 1949.

injected systemically and into the anterior chamber. Such a case is reported by Shoemaker.[139] In his case, the organism was more sensitive to penicillin than to streptomycin; yet the administration of penicillin systemically and its injection into the anterior chamber were ineffective. On the other hand, two direct injections of streptomycin into the vitreous for a total of 2,300 micrograms resulted in subsidence of the infection, and the final visual acuity was 20/200.

Von Sallmann,[140] after a careful investigation of the effects of the intravitreal injection of penicillin, concluded that the eye could safely tolerate between 1,000 to 2,000 units.

Mejer [141] treated 12 patients with post-traumatic or postoperative infections of the vitreous by the creation of a vitreous fistula combined with the injection of streptomycin and penicillin subconjunctivally, thus achieving high concentrations of the antibiotics. In five patients good results were obtained. The poor results in the other seven were attributed to the presence of resistant bacteria, unrecognized infections of the lens and delayed treatment.

Pathologic Features.—An interesting ocular manifestation of the uveitis caused by sarcoidosis which occurred in three young Negro patients is described by Landers.[142] In the dependent portion of the vitreous, he observed discrete, grayish white, spherical or tubular bodies, which persisted even after the uveitis had become quiescent. He postulates that these bodies form either from collections of lymphoid cells or from serous exudates.

Cassady [143] reports the occurrence of a congenital cyst of the vitreous in a 9 year old boy. The cyst was round and floated in the vitreous with the ocular movements but returned to its central position when the eye came to rest.

Vitreous Hemorrhage.—Radnot [144] discusses the pathogenesis of hemorrhages into the vitreous humor. He agrees with the older writers who believed that the disease is based on periphlebitis retinalis tuberculosa. He mentions the fact that Marchesanis, disagreeing with this opinion of the cause, believes the hemorrhages are due to thrombotic changes in the retinal vessels. An important contributory factor may be a disturbance of internal secretion.

Cysticercus of the Vitreous.—Involvement of the eye with the larva of Tenia solium is known as ocular cysticercosis. Extraocular cysticercosis is relatively infrequent, probably because the parasite is well tolerated and the disturbance, which is noted early when the eye is involved, is insufficient to make the patient aware of it in other parts of the body. The vitreous body is the ocular tissue most commonly affected. The lens is never involved.

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139. Shoemaker, R. E.: Intravitreal Use of Streptomycin, Arch. Ophth. **41**:629-632 (May) 1949.

140. von Sallmann, L.: Value of the Intravitreal Administration of Penicillin, Am. J. Ophth. **32**:580-582, 1949.

141. Mejer, F.: Zur Therapie exogener Glaskörperinfektionen, Klin. Monatsbl. Augenh. **115**:384-390, 1949.

142. Landers, P. H.: Vitreous Lesions Observed in Boeck's Sarcoid, Am. J. Ophth. **32**: 1740-1741, 1949.

143. Cassady, J. V.: Congenital Cyst of the Vitreous, Arch. Ophth. **41**:734-735 (June) 1949.

144. Radnot, M.: Beitrag zur Pathogenese der rezidivierenden Glaskörperblutungen, Klin. Monatsbl. Augenh. **115**:327-332, 1949.

Swett [145] reports a case of cysticercosis of the vitreous in a Mexican. He found that each sucker contained an extendible tubule, not previously mentioned. Junior [146] reports from Brazil that 111 cases were observed among 153,528 patients. The diagnosis is made by the identification of a bluish white, iridescent, spherical vesicle which, at times, shows undulating movements. The scolex when evaginated shows sucking cups, and when invaginated appears as a dense white spot in the center of the vesicle. Complement fixation is positive in over 70 per cent of cases. The living and the dead parasite are equally destructive to the globe and must be extracted if the eye is to be preserved. The author states that prophylaxis is effective and consists in the control and inspection of meat and in personal dietary hygiene.

30 North Michigan Blvd.

145. Swett, W. F.: Cysticercus of the Vitreous, Am. J. Ophth. **32**:574-577, 1949.

146. Junior, L.: Ocular Cysticercosis, Am. J. Ophth. **32**:523-548, 1949.

Correspondence

THE HEED OPHTHALMIC FOUNDATION

Report of Activities from Inception in May 1946 Until October 1950

To the Editor:—The Heed Ophthalmic Foundation was conceived by Mr. Thomas D. Heed, of Chicago, and his wife, Ruth Heed, in an effort to give additional opportunities for surgical experience and study to young ophthalmologists of exceptional ability. They felt that frequently the contribution that such men might be able to make to the advancement of the science of ophthalmology was lost, either through the pressure of finances or from the lack of entrée to the best institutions of the nation. It was their belief that many deserving young men who had married or incurred other obligations on the completion of the usual curriculum, though such actions were eminently proper and right, were thereby forced to abandon their studies prematurely for the more lucrative field of clinical ophthalmology. Therefore, after consultation with leading ophthalmologists from various parts of the country, a trust agreement was drawn up, and five directors were appointed to carry out the details of the program.

In the beginning, the possibility of establishing surgical Fellowships was investigated, and letters were written to 12 of the major clinics of the country, seeking such Fellowships. The replies, however, soon made it evident that it was not practical for the various university clinics to grant surgical opportunities to men other than those in training in their own institutions, no matter how exceptional might be the ability of such men. The plan, therefore, had to be altered. The founders readily agreed to all suggestions made by the Board of Directors and gave up the idea of surgical Fellowships, accepting in their stead the arrangement which is now in operation.

The procedure is very flexible and permits great latitude on the part of the Board of Directors. The usual program is based on a six month period, which may be prolonged to one year, or more, as may seem desirable. The time occupied by the usual six month period is divided between three situations, no less than two months being spent at any one station. It has been felt that any shorter stay precluded the Fellow from becoming an integral part of the organization, and held him in the role of a visitor, so to speak, rather than a temporary member of the staff. In each clinic the work is assigned under the direct supervision of the chief of staff. Thus far, the Fellows have been afforded the opportunity of attending all lectures, demonstrations and surgical procedures in the same manner as the regular residents of the institution, with similar privileges, except those of performing surgical operations. In a few instances, such operative privileges have been granted to a limited extent. In addition, the Fellows have also been encouraged to undertake simple research problems, though the time limits are such as to preclude elaborate programs of this nature, even though the problem may be carried from one clinic to the next.

In exceptional cases, other courses of study have been offered. One Fellow spent six months working in histopathology at one institution, and was granted an additional six months of similar study at another station. At the present time, one candidate is under consideration for special training in the management of problems in ocular motility. Thus, it can be seen that the Foundation is carrying out the ideas of the founders in helping men who give promise of exceptional ability to continue their studies along general or special lines of work on a very liberal basis.

Up to the present time, two men have had one year's training, and six others have had six months. In one instance withdrawal was forced, because of illness in the family, at the end of the first two months. At the present time there are three Fellows in the midst of their training. All those thus far accepted have proved to be men of outstanding ability and are giving such promise that the founders and directors of the Foundation have felt great satisfaction with what has been accomplished.

Great credit should be given to the various chiefs of clinic, their staffs and their institutions who have assisted in this program. Among such should be mentioned the Department of Ophthalmology of the University of California Medical School; the Department of Ophthalmology of Columbia University College of Physicians and Surgeons; the Eye Bank Laboratory of the Manhattan Eye, Ear and Throat Hospital; the Department of Ophthalmology of Johns Hopkins University School of Medicine; the Massachusetts Eye and Ear Infirmary; the Department of Ophthalmology of Northwestern University Medical School; the Department of Ophthalmology of the University of Oregon Medical School.

As in all other matters pertaining to the management of the Fellowships, the founders have left the details concerning the stipend to the discretion of the Board of Directors. The amount has been established with a view to rendering assistance to a number of candidates, rather than concentrating too highly on one or two at a time. An effort has been made to adjust the remuneration at such a level that applicants will look on the compensation not as a complete livelihood but, rather, as an assistance in carrying on work which they greatly desire. The idea has been that, established on its present basis, it will attract those who realize the rare opportunity which is granted them of access, on the most intimate and instructive basis, to the finest teachers and clinics in the country.

There has been no effort to solicit the name of any candidate, other than to let it be known from time to time that one or more Fellowships were open to those having the necessary qualifications.

In addition to the exceptional qualifications outlined above, it is required that all candidates be citizens of the United States. Following out this stipulation of the trust agreement, it has been necessary to refuse Fellowships to several most desirable candidates, but should they have been accepted, equally fine men of American birth would have been denied the opportunities offered.

It was Dr. Harry S. Gradle who first interested the founders in the possibility of advanced education in research and surgery for young ophthalmologists of exceptional ability.

M. Hayward Post, M.D., Secretary.

News and Notes

EDITED BY DR. W. L. BENEDICT

SOCIETY NEWS

Eye, Ear, Nose and Throat Hospital (New Orleans) Alumni Association.—The first annual meeting of the association will be held in New Orleans, April 26 to 28, 1951. All physicians who have received their specialty training at this hospital are cordially invited to attend.

One day will be devoted to ophthalmology and another to otolaryngology. On the morning of April 28 there will be demonstrations of a number of operative procedures in each specialty.

Each night there will be a cocktail party. During the day the wives of the alumni will be entertained at tea, taken on tours of homes in New Orleans' famed French Quarter and Garden District and taken on a yacht trip on the Mississippi River, during which a buffet luncheon will be served.

Inquires should be addressed to George H. Hauser, M.D., 145 Elk Place, New Orleans.

Obituaries

WILHELM H. BUSCHKE, M.D.
1907-1950

The sad death of Dr. Wilhelm H. Buschke has deprived the science of ophthalmology of an extremely competent investigator and has cut short an extraordinarily productive career of great promise and achievement. Dr. Buschke was born in Berlin in November 1907, the son of the distinguished dermatologist Dr. Abraham Buschke and of Erna Buschke. He was the youngest of three brothers, of whom the oldest, a radiologist, has also achieved distinction in medical research.

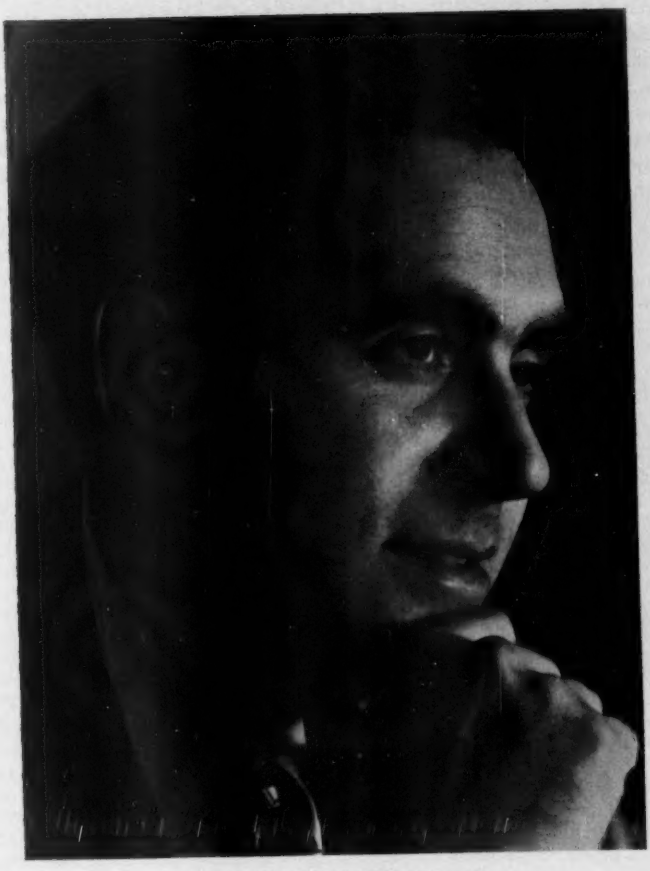
Wilhelm Buschke obtained his elementary education in Berlin and his premedical and medical training at the Universities of Freiburg and Berlin, receiving his degree of Doctor of Medicine in 1932. His training in ophthalmology and his induction into ophthalmologic research was chiefly under the guidance of Prof. Hans Goldmann, in Basel and Berne, where he worked from 1932 to 1937. He was research assistant in the University Eye Clinic in Berne from 1936 to 1937. Many of his studies during this period were done in collaboration with H. K. Müller and were concerned with the role of ascorbic acid in lens metabolism. He was deeply devoted to Professor Goldmann and often spoke with glowing enthusiasm about the penetrating scientific insight and personal kindness of his teacher.

In 1938 he came to the United States and worked as research assistant and instructor in ophthalmology at the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and Medical School from 1938 to 1947. In March 1947 he became research ophthalmologist to the Manhattan Eye, Ear and Throat Hospital in charge of the Ayer Foundation Ophthalmic Research Laboratory, which post he held up to the time of his death.

The period of his work in Baltimore was one of intimate and happy collaboration with him for myself and for the other members of the Wilmer Institute staff. His work here began as a continuation of his studies on ascorbic acid, leading to disclosures of the role of ascorbic acid, and later of epinephrine, in the mechanism of secretion of the intraocular fluid. He played an active part in the studies on ocular injuries by toxic war gases undertaken at the Institute during World War II and extended the field of his studies into problems of cataractogenesis and of mitosis and wound healing in the corneal epithelium.

Always he brought to his problems a wide scholarship and a high degree of originality both in outlook and in the devising of simple and novel technics. As time went on, he became especially interested in the role of the divalent cations, particularly calcium and magnesium, in the maintenance of tissue structure and organization and developed plans to apply to ophthalmologic problems the insights and findings of the physiologist Heilbrunn in this field.

It was thus, as a disciple of Heilbrunn, that he began his work in the Ayer Laboratory, when he focused his attention on problems of cellular cohesion in the corneal epithelium and on cytoplasmic sol-gel transformations, and on the role of the divalent cations in these recondite processes. Even though his studies in



WILHELM H. BUSCHKE, M.D.
1907-1950

this field were interrupted so soon by his untimely death, his publications during this period showed that he had found a richly rewarding domain of scientific inquiry, in which there were potentially important harvests both for the elucidation of basic physiological and pathological mechanisms and for significant clinical applications.

His brief career has left a deep imprint on the minds of those engaged in research in fields allied to his own. In the short period of less than two decades he published over 60 papers, all bearing the pattern of scholarship, meticulous scientific care and insight which marked his work.

To those who knew him, his untimely death constitutes a deep personal loss, for his personal relations were characterized by gentleness, good humor and kindness, which enriched the lives of those who came close to him. Always profoundly concerned with what was true and right, he struggled, often with inner anguish, but always with grace and humility toward his fellow man.

JONAS S. FRIEDENWALD, M.D.

Book Reviews

Current Therapy, 1951. Howard F. Conn, Editor. Price, $10. Pp. 699. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 3, 1951.

For the third time, this annual outline of therapy has appeared on the market, written by a wide variety of experts under the editorship of Howard F. Conn. Although there is no section on the eye itself, various conditions of the lids, such as allergy of the skin of the lids, seborrheic dermatitis, xanthoma and other local conditions, are given consideration.

The main value to the ophthalmologist will be to keep abreast of changes in therapy of general diseases, and for this the book can be heartily recommended.

Vernal Conjunctivitis. By M. N. Beigelman, M.D. Price, $6. Pp. 430, with 49 figures. University of California Press, California Hall, Berkeley 4, Calif., 1950.

Dr. Beigelman's monograph on vernal conjunctivitis is an extensive critical review of the literature, evaluated on the basis of the author's experience with 236 cases of vernal conjunctivitis. Beginning with a discussion of nomenclature, the history of the disease is presented in three periods. The first period begins with Arlt's remarks in 1846 and extends to Brockhaus' presentation of his and Saemisch's contribution in 1872. The second period extends to the time Axenfeld presented his critical review of all work on vernal conjunctivitis before the twenty-fourth conference of the French Ophthalmological Society in 1907, and the third period extends to the present time. Perhaps Beigelman's critical review will mark the end of the third period. In the first period the disease was established as a clinical entity by Arlt, Desmarres and von Graefe. Saemisch recognized the periodicity of the disease, which he called spring catarrh. In the second period the clinical aspects were studied in more detail, and, finally, in the third period the distribution of the disease throughout the world was emphasized. Beigelman cites the contributions of Herbert, who discovered the eosinophilia in the conjunctival secretion of vernal conjunctivitis, in 1903; Barnett, who described the abnormal pigmentation of the conjunctiva, in 1881; Gradle, who recognized the similarity of vernal conjunctivitis to hay fever, in 1886, and May, who discovered the possible coexistence of vernal conjunctivitis and trachoma, all of which features were overlooked for many years.

Clinical aspects of the disease are presented, both by discussion and by tabulation of reported data. In summarizing the data on sex incidence, the author uses only series of more than 50 cases, but 2,447 of 3,172 cases occurred in males. Although vernal conjunctivitis is essentially a disease of youth, all but approximately 5 per cent of cases occurring before the age of 30, a 74 year old and a 75 year old patient have been reported. All races are subject to attacks, and there seems to be a familial tendency; but a detailed analysis of hereditary factors is not yet possible. Because of a number of sources of statistical error, the data on regional variations of the disease are difficult to analyze; but in general the hotter regions of the world show a higher incidence of the disease, and likewise a higher incidence of limbic and mixed types. Subjective symptoms consist of itching, foreign body sensation, burning, lacrimation and photophobia; but itching is by far the commonest complaint.

Although the practical classification of vernal conjunctivitis by Emmert divides the disease into palpebral, limbic and mixed types, Trantas, Weskamp and Puig Solanes insist that rudimentary, corneal and atypical forms occur and should be recognized.

Changes in the tarsal conjunctiva include diffuse tissue hyperplasia, loss of luster, discoloration, papillary hypertrophy, large excrescences, ptosis and pseudoptosis. Pericorneal changes are characterized by dulness and opacification of the limbus, followed by thickening, especially in areas of the palpebral fissures, or pericorneal vegetations, which may contain "white points," as described by Horner and Trantas; and, finally, pits may appear as foci of regression within the hyperplastic formations. The scleral conjunctiva in many instances remains normal, but not infrequently it loses its transparency and elasticity. Occasionally tissue proliferation produces a "goose skin" appearance of the conjunctiva. Vascular changes, plaques, nodules, polyps and pseudopterygium occur. Characteristic brownish discoloration of the limbal excrescences are well known, but occasionally pigmentation of the scleral conjunctiva occurs, particularly in dark-skinned persons.

Corneal involvement consists of lesions due to direct continuation of the conjunctival disease into the cornea, lesions without direct connection with the affected limbus and dystrophies of the cornea. Among the lesions due to direct continuation of the conjunctival disease are advancement of the opaque limbal zone, pannus-like formations, nodular hyperplasia and sclerocorneal proliferations. Corneal lesions without direct connection with the affected limbus consist of keratitis epithelialis, subepithelial punctate lesions, deep keratitis and corneal ulcer. Dystrophies of the cornea include pseudogerontotoxon; changes in the corneal curvature, resulting in myopic astigmatism; keratoconus, and keratoglobus.

The author describes the appearance of these manifestations both from the gross and from the biomicroscopic standpoint.

Although self limited, the number of seasonal recurrences in the course of the disease fluctuates widely. Usually the disease runs a course of four to six years; but Uhthoff reports a case of 35 years' duration, and Trantas, one of 29 years' duration.

The secretion is usually scanty, stringy and sticky. Occasionally the discharge may be copious. Herbert observed the formation of a fibrinous pseudomembrane on the conjunctival surface of the everted lid. He also reported numerous eosinophils in the membrane and in the secretions.

Studies of the bacterial flora have not revealed any organism associated with the disease. Inclusion bodies have not been found consistently and are coincidental.

The author gives a detailed differentiation of the disease from trachoma, phlyctenular conjunctivitis, infectious granuloma and neoplasms, as well as various types of conjunctival allergy. He discusses the relation of the disease to vagotonia, status thymicolymphaticus and endocrine disturbances. He gives detailed descriptions of the histopathological changes and correlates these with the clinical manifestations.

From the standpoint of etiology, the author discusses constitutional factors; climatic and physical factors, such as temperature, air humidity, proximity to large bodies of water, altitude, air movement and radiant energy; exogenous infectious agents; generalized infectious diseases; photosensitivity, and allergy. He concludes that hypersensitiveness, mainly to material substances, must be considered the most promising hypothesis.

In the final division of the book, under therapy, the author discusses practically all therapeutic measures and drugs that have been employed up to the time of the introduction of the antihistaminics. This, of course, excludes the use of pituitary adrenocorticosterone (ACTH) and cortisone. Thus, it would seem to the reviewer that Beigelman's volume ends the third historical period of the disease. It forms a worthy conclusion to the period begun by Axenfeld's contribution. It will be an essential volume in the library of all men profoundly interested in the disease or in external diseases of the eye.

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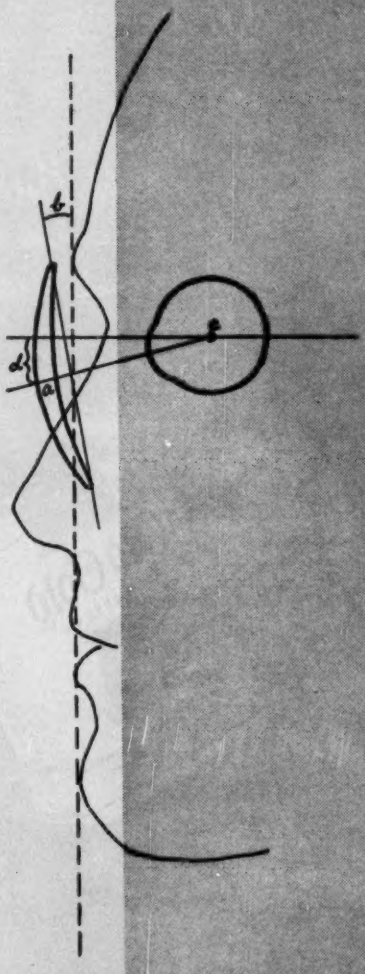

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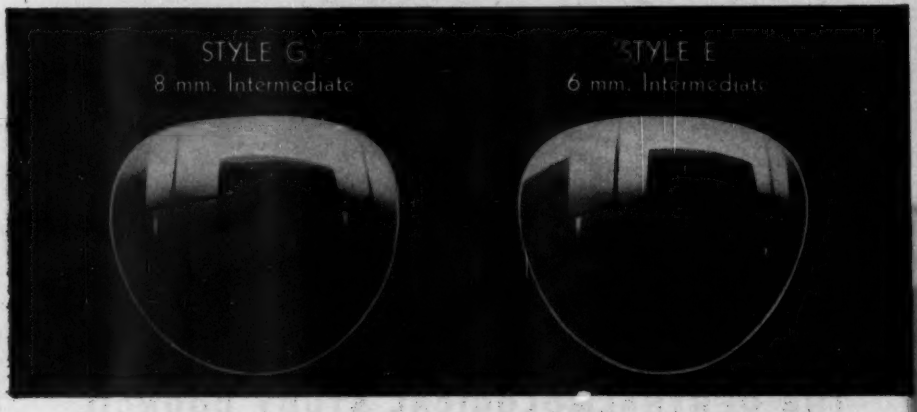

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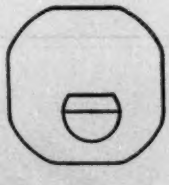

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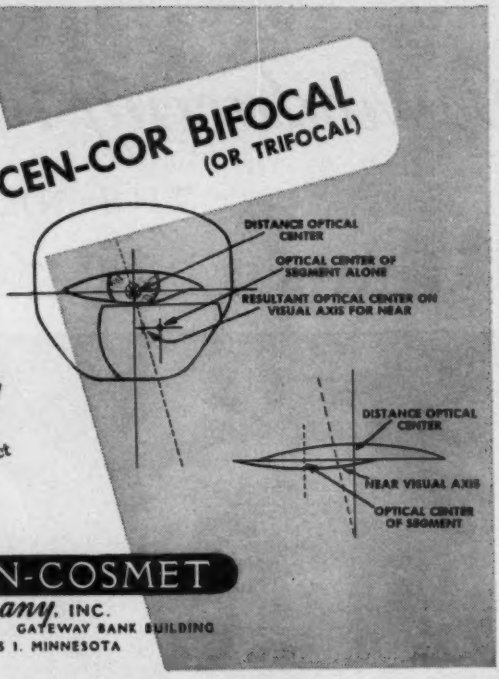
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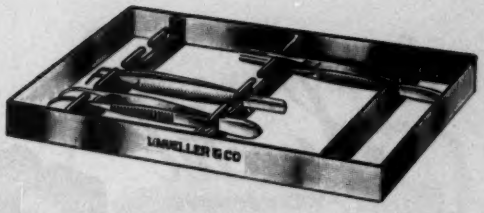

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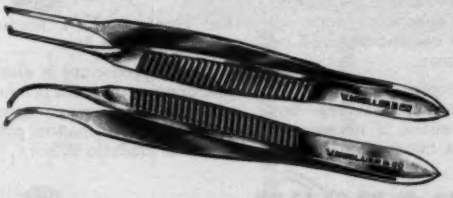

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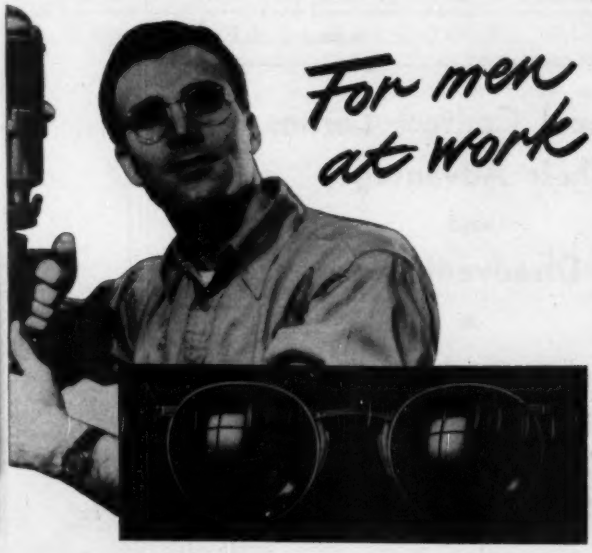

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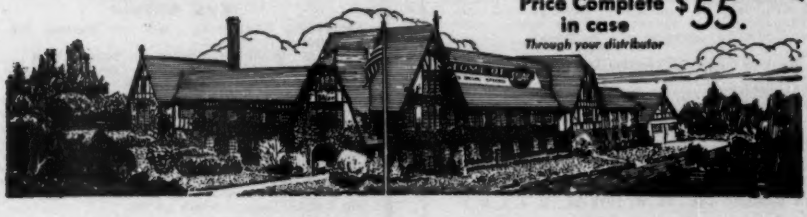

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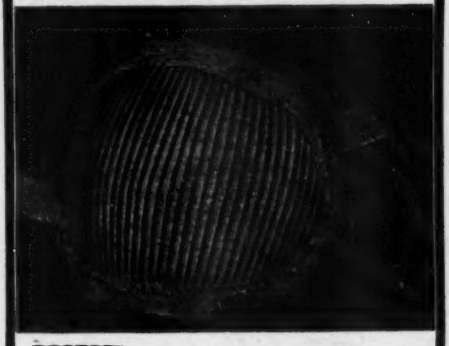

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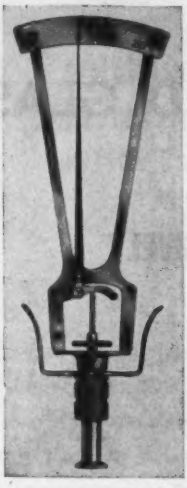

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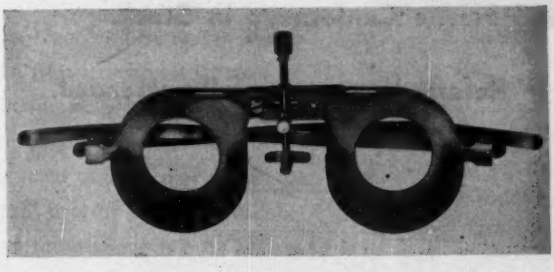

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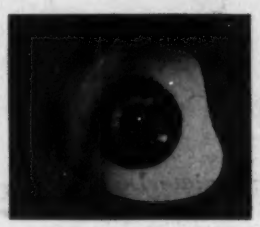

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For short acting powerful mydriatic effect, applied 30 to 60 minutes before surgery.

Uveitis, posterior synechiae: **10% solution and 10% emulsion.**
For freeing recently formed posterior synechiae as well as for prevention of synechias formation in uveitis (with atropine).

Glaucoma (certain cases and tests): **10% solution, 10% emulsion and 2.5% solution.**
For the provocative test for angle block as well as for the shadow test.

NEO-SYNEPHRINE®
HYDROCHLORIDE
Brand of
Phenylephrine hydrochloride

Winthrop-Stearns Inc.
New York 13, N. Y. Windsor, Ont.